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ARCHIVES OF OPHTHALMOLOGY.

I. THE PRIMARY PUPILLARY RESPONSE TO LIGHT AN AUTONOMIC REFLEX.

II. THE ARGYLL-ROBERTSON PUPIL.

BY JOHN DUNN, M.D., RICHMOND, VIRGINIA.

FOR many years the problem of the Argyll-Robertson pupil has interested me and my efforts to solve it finally resulted in the belief that the long and universally accepted views of the primary light reflex are incorrect. My reasons for this belief are stated below. The explanation here given is original with me, although I make no claim that similar views may not have been held before. If so, however, I am unacquainted with them. What exactly is meant by the Argyll-Robertson pupil? It is, I quote De Schweinitz: "A pupil which does not react directly or consensually to the influence of light, but contracts promptly on convergence of the visual axes." This is the accepted definition. We shall see later on that it does not bring out an essential feature and one whose recognition is necessary to the full appreciation of the phenomenon—How is the Argyll-Robertson pupil explained? Where is the seat of the lesion which gives rise to this reflex iridoplegia? Starr, *Nervous Diseases*, p. 354, says: "The mechanism of this reflex is still a matter of dispute, and it is not definitely settled whether the break in the reflex arc lies in the segment between the second and third nerves near the corpora quadrigemina or is through the sympathetic nerve which has its origin at the first dorsal segment of the cord." On p. 638 he says:

"There is as yet no satisfactory explanation for the occurrence of this symptom in tabes." And in this connection he says the Argyll–Robertson pupil "occurs in tabes, paresis, in disseminated sclerosis, in softening or atrophy of the brain, in hydrocephalus, in tumors of the third ventricle or adjacent basal ganglia, and as a congenital condition." I quote Starr because of the justly high position his work on *Nervous Diseases* is entitled to and to show how entirely wrong my explanation is, if the pure type of the Argyll–Robertson pupil can be produced by lesions in the above-mentioned conditions. Weeks, *Diseases of the Eye*, p. 122, says the Argyll–Robertson pupil is "due to the destruction of the fibers that pass along the optic nerve, before they reach the third nerve." I am unable to find that he states anything more definite as to the seat of the lesion. Such a statement of the case is most unsatisfactory. De Schweinitz, *Diseases of the Eye*, p. 76, says: "The seat of the lesion is not certainly known. It has been placed in the fibers which pass from the proximal end of the optic nerve to the oculomotor nuclei by some authors, and by others it is considered to be nuclear. Bach believes it may be located in the spinal end of the sinus rhomboidalis and Marina and Lafon place it in the ciliary ganglion." I am extremely sorry never to have seen Marina's and Lafon's communications on this subject, and I do not know what their explanations of their belief are. Bumke, in one of those marvels of Teutonic thoroughness, after pages of discussion on the Argyll-Robertson pupil says—I give the translation: "There remain only two possibilities: either reflex iridoplegia is the result of a disease in the nucleus for the motor innervation of the sphincter iridis, or is the result of a change which leaves intact both the sensory and motor reflex fibers and hinders the transmission from the one to the other." Bumke does not agree that the lesion can be in the ciliary ganglion. He believes that every requirement of the explanation is to be found in a lesion which breaks the reflex centripetal fibers in the region of the sphincter nucleus. He ends, howevre, his defense of his position with the very significant statement that the exact determination of the pupillary reflex tract is not only the next but the most important thing to be determined. To my mind this means that he is not at all satisfied with his explanation.

The pupillary reflex tract—What is it? One would imagine that there is nothing in all Ophthalmology so fully determined as the path along which the light impulses from the retina pass to result in contraction of the pupil. For my own part until I began to try to solve the mystery of the Argyll-Robertson pupil, and this was a good many years ago, I unquestioningly accepted the statements of the physiologies and the ophthalmological text-books. It never once occurred to me that their views were not beyond question. How I ever began to doubt their accuracy I do not know.

Weeks, opposite p. 120, gives a beautiful diagram to show exactly the pupillary reflex light arc, and in this diagram is summed up the accepted belief, so far as I know, of medical science on this subject. De Schweinitz, *Diseases of the Eye*, seventh edition, p. 171, states it as follows: "In as much as the iris is not under the control of the will, the contraction of the pupil which occurs when the eye is exposed to the source of light in the manner described is a reflex—that is, its motor nerves are excited to action indirectly by the reflex stimulus of light. The light reflex is under control of the constrictor center which the stimuli reach by passing along a tract which is known as the *afferent pathway*, the exact course of which is uncertain but is probably somewhat as follows: the fibers of the pupil-reflex tract begin in the retina and arise from all parts of it and proceed in the optic nerves and are in all probability to be histologically differentiated from those which are concerned with vision. In the chiasm these pupillary fibers undergo partial decussation and enter the optic tracts, which they leave just in advance of the external (lateral) geniculate body and reach the third-nerve nucleus. From a special part of this nucleus, probably the small-celled median nucleus, the pupillo-constrictor fibers arise and reach the sphincter of the iris, constituting the afferent, myotic, or centrifugal pathway already described. (The path by which the pupillary fibers leave the optic tract to reach the third nucleus, in the language of Parsons, is as yet conjectural. The author, referring to the pupillo-constrictor path, thinks it probable that the fibers pass through the superior brachium of the quadrigeminal body to the superior colliculus, there making new connections with the cells which carry the impulses

to the third-nerve nucleus of the same and opposite side.) *The direct light reflex of the pupil is the result of an active constrictor effect*, the stimuli passing along the afferent pathway to the sphincter center in the third nucleus, and from there by the efferent pathway to the termination of the myotic fibers in the iris."

De Schweinitz adds: "Marina, instead of locating the center for the light reflex of the pupil in the small-celled nuclei which occupy the median part of the third nucleus, places it in the ciliary ganglion." Again, I have not seen Marina's reasons for his belief.

We must now ask a few questions. Why should there be so elaborate a bit of mechanism as the one described above, and generally accepted, for the production of so simple an unconscious act as the contraction of the sphincter iridis?—an act that occurs in a healthy eye without any reference to anything the contraction may affect. In the pupillary response to light we are dealing with the simplest type of a reflex—and we should expect nature to make use of the simplest type of mechanism capable of producing such a reflex. As the brain is incapable in any way of preventing the accomplishment of this reflex when the conditions for its production are present, we should again expect nature not to put upon the brain cells an unnecessary burden, provided, of course, its accomplishment is possible without recourse to the action of the higher nervous centers. This brings us to our next question: Is there any path by which the contraction of the sphincter can be brought about as the result of the impact of light on the retina which does not pass into the central brain substance? If so, what is it? We know that sympathetic, motor, and sensory nerve fibers pass into the back of the eye from the ciliary ganglion. Why should not some of these sensory nerves have some connection with the retina and be responsive to light influences? And right here is the seeming impasse which has shunted off all ophthalmologists, so far as I know, save Marina and Lafon, down the apparently easier route of the optic nerves—a route which, so far as our problem is concerned, has led them no whither. The anatomists tell us "there are no fibers carrying common sensation connected with the retina" (Piersol). If this be true, why has nature

continued that part of the retina so wonderfully responsive to light impulses—the pigment layer—to both the ciliary and iritic regions, to both of which are bountifully supplied sensory fibers from the ciliary ganglion? It must be for some purpose.

When light falls upon the retina, marked activity is aroused in the cells of the pigment layer, the granules of which advance and retreat along the protoplasmic processes enveloping, to a degree varying with the intensity of the light, the outer membranes of the visual cells. It is inconceivable to my mind that this activity of the pigment cells should cease with the exact limitations of the retina, and I believe that this activity is continued along the pigment layer to the region of the ciliary bodies, reaches and is responded to by the sensory elements of the short ciliary nerves and is transmitted by them to the ciliary ganglion. If this be true, then we have the route for the direct response of the ganglion to light impressions, *i.e.*, a response which brings into play the motor fibers to the iris without the intervention of the brain centers. Such a route I believe to be necessary to an explanation of many iritic phenomena.

The route then for the pupillary light reflex, instead of being the intricate thing of the text-books, would be as follows: *Retina, nerve cells to the pigment layer, and along this to the ciliary region where impressions are made upon the sensory nerve terminals therein, which impressions are conveyed to the ciliary ganglion where they arouse efferent impulses along its motor fibers to the sphincter pupillæ.* This, I would term *the primary light reflex* and, as I see it, the appreciation of what constitutes a primary reflex opens the way to the understanding of the *raison d'être* of the whole autonomic system and makes possible the interpretation of many of the now unintelligible symptoms referred correctly to the domain of the extended vagus, and allows us to do more than guess at the foci of selection of the drugs used in our experiments to determine the degree of stability of the vagus tone. Indeed it opens the door for the entry of many suggestions explanatory of important well-known but not understood "nervous symptoms." In short, the primary light response on the part of the sphincter is a vegetative reflex. Dr. Brubacher, of Philadelphia, in the *Annals of O. T. and L.*, September, 1916, in an article on the

"Innervation of the Nasal Mucosa," states of the sphenopalatine ganglion cells that "though irritable they are devoid of spontaneity of action and *require for their excitation the arrival of nerve impulses, discharged by the nerve cells in the central nervous system and transmitted by the preganglionic fibers.*" (Italics mine.) Apply this belief to other extracerebral ganglia, in especial to the ciliary ganglia, and I believe the Argyll–Robertson pupil would be forever incapable of explanation. My own belief in regard to all the extracerebral ganglia, and this applies to all the ganglia of the so-called extended vagus, is that *they are capable of response from appropriate irritants applied to the terminations of the sensory fibers from these ganglia as long as they are normal and the preganglionic fibers are intact.* It is this property that makes possible the primary reflex. In the ciliary ganglion it makes possible the primary light reflex. Into the ciliary ganglion enter sensory fibers from the fifth nerve, fibers from the sympathetic, and motor fibers from the third nerve. What transformations and connections these various preganglionic fibers undergo within the ciliary ganglion have, so far as I know, not been exactly worked out. They form however, extracerebral connections of such a sort that not only can impulses from the preganglionic fibers pass through the ganglia and regulate its action, but also of such a sort that from the periphery sensory impulses can originate within the ganglion motor impulses which are manifested as primary reflexes. The necessity for such reflexes in the domain of the vegetative system is evident. These primary reflexes can be regulated by the higher centers; they cannot be prevented. They are the momentary checkreins which hold until a guiding hand gains control. *It is the abolition of this primary reflex of the ciliary ganglion which gives us the Argyll–Robertson pupil.*

The second fact in connection with the Argyll–Robertson pupil is found in the statement that "the consensual light reflex is also abolished." When both eyes are normal, no matter what the difference of illumination before the two eyes, the two pupils remain of practically the same size—this is the result of what is called the consensual light reflex. The exact variations of this reflex are beyond our discussion here. What is the path for this reflex? And how comes it that it, too,

seems to be abolished in the Argyll-Robertson pupil? How can a lesion in the ciliary ganglia abolish a reflex which so manifestly must have a more complex path than that of the simple light reflex? *It is not abolished. It is not called into play*, because the two pupils, not being responsive to light, do not change their size. What is the need for the consensual reflex? Why should there be one at all? Why did nature establish it as a part of the act of vision? Why should the pupils be kept of the same size? These questions need not be elaborated here—the answer in brief is—its necessity grew out of the fact that at no given moment is the amount of light entering the two eyes ever the same. You can prove this by closing first one eye and then the other—and from the further fact that *experience has taught the brain that no single object can reflect two different degrees of illumination at the same instant.* The consensual light reflex, then, is the result of nature's effort to obtain for any object directly looked at a like illumination for both eyes. This is interesting in that it gives to our conception of the function of the optic nerves a breadth which, if heretofore recognized, is not dwelt upon in the text-books. It throws a new light upon the interpretation of intracranial lesions which affect both the sight and the pupil. It shows the vast importance of the consensual reflex, and, finally, it explains the necessity for the double cerebral origin of the optic nerves; in other words it explains why the optic nerve originates from "both the stratum opticum of the corpora quadrigemina and from the optic thalamus," making close connection with, if not originating from, that portion of it known as the subthalamic sympathetic center, which corresponds to the median part of the frontal portion of the thalamus—the center of Karplus and Kreidl. What then is the path for the consensual light reflex? The retina, the optic nerves, the chiasm, and tracts to both of their origins. Why to both origins? Because it is necessary to awaken two kinds of impulses—constrictor and dilator. We are no longer dealing with a simple, a primary reflex. We here have to do with a reflex accurately regulated for a definite purpose. A portion of the optic-tract fibers, a part from each retina, go to their centers in the geniculate and quadrigeminal regions. From these centers pass fibers to the third-nerve nuclei. From these

centers proceed fibers for the optic radiations. Further details of this I must omit here. The other fibers from the retina proceed along the optic nerves, chiasm, and tracts to the two sympathetic subthalamic ganglia. Their function is to stimulate the action of the sympathetic on the dilatator iridis. Thus it comes about that we have a mechanism provided for an equal sympathetic action at all times on the dilatator fibers of the two eyes. I shall repeat a part of this as it clears up the point as to how nature provides for the consensual reflex of both the sphincter iridis and the dilatator iridis, and, furthermore, because it makes a suggestion, new to me, as to why the retina contains both rods and cones. It is possible that the cones represent the terminal mechanism for the reception of the impulses to the geniculate and quadrigeminal nuclei; and the rods, of the impulses to the subthalamic sympathetic ganglia. The chiasm serves to make possible an equal stimulation, not only of the quadrigeminal centers, *i.e.* those centers which are directly concerned in vision, but also an equal stimulation of the subthalamic sympathetic centers, which with the third-nerve centers are concerned in the mechanical adjustment of the focus and the proper illumination of its field. The long and complicated route for the sympathetic dilatator impulses from the subthalamic ganglia to the ciliary nerves and by these to the dilatator iridis has been fairly well worked out and I shall omit comment upon it, save to say that the recognition of the fact that about half of the fibers in the optic nerves are in connection with each subthalamic ganglion impresses us with the importance of this ganglion. The further recognition of the extracerebral ganglionic connections that the fibers from the subthalamic ganglia make, on their route to the dilatator iridis, helps greatly in the understanding of the many phenomena of pupillary dilatation which occur independently of retinal impulses. Now to return to the Argyll–Robertson pupil. We have seen the seat of the lesion which makes possible reflex iridoplegia. We see that this lesion does not do away with the consensual reflex—but that this reflex is *absent because there is no call for its manifestation, there being no change in the size of the pupils.* The third element of the Argyll–Robertson phenomenon is that the pupil responds in convergence and in

accommodation. If the lesion be situated, as I think, in the ciliary ganglia, and if the lesion be confined to the sensory elements, leaving the motor element still connecting responsive to all impulses which reach it from the third-nerve centers, then there is no reason why the iris should not contract in convergence and accommodation, and, further, no reason why the consensual light reflex should not continue in force as a part of the convergence and accommodative acts—as under normal conditions. This is the fact to which I had reference when I said above that no previous description of the Argyll-Robertson pupil is complete. In considering the ciliary ganglion it may be well to stop for a moment to ask what we know about its functions. Paralyze the third-nerve fibers to the ciliary ganglion, either by a lesion in the nuclei of origin or by section of the third nerve anywhere posterior to the ganglion, and we produce dilatation of the pupil. It is characteristic of this dilatation, however, that it is not *ad maximum*. Destroy the ciliary ganglion or the short ciliary nerves emerging from it and we at once get complete dilatation. Furthermore, the dilated pupil in both instances is irresponsive to light. It seems fair to deduce from these facts that there resides within the ciliary ganglia a control over the pupil above and beyond that exercised by the third-nerve nuclear centers. We see, too, that the pupil deprived of the impulses which reach it from the sphincter pupillæ fibers, even though the ciliary ganglion be intact, no longer responds to light impulses, either direct or consensual. This shows that the impulses derived from the nuclear station are necessary for movements of the pupil and yet complete paralysis of the sphincter cannot be obtained by paralyzing the central nuclei so long as the ciliary ganglia and their peripheral connections remain unimpaired. The difference between the sizes of the pupils in the two types of paralytic dilatation under consideration may be taken roughly as a measure of the power for pupillary contraction residing in the ciliary ganglion. That any power for contraction of the pupil is left after the abolition of the central impulses to the ganglia shows clearly that there are two separate forces at work in the contraction of the pupil. It shows, too, to my mind, that motor fibrils pass directly through the ganglionic substances, even though within the

ganglionic cells connections are made with the sensory and sympathetic cells. It shows, further, that the consensual, and not the primary, reflex is the great purpose of the sphincter pupillæ response. Normally the pupil responds to light, but for the higher purposes of the brain this response needs the regulation which is expressed in the consensual reflex. Remove the possibility of the production of this reflex and there is relatively so little use for the primary reflex that it is not called into play. Had this provision for the abolition of the primary pupillary reflex following the abolition of the secondary, the consensual, reflex not been made, we should have unregulated sphincter responses to light impulses and the pupillary movements would be a veritable *lusus naturæ*. I believe all the primary reflexes born of the extracerebral ganglia are subject to a like provision. This brings us to the point where we must answer a few of the questions which doubtless have suggested themselves to those who have ever undertaken to explain the Argyll–Robertson puzzle. If the primary reflex response to light impulses is abolished, why in the typical Argyll–Robertson phenomenon are both pupils contracted—frequently extremely so—so much so, in fact, that no further contraction in the acts of convergence and accommodation are demonstrable to the unaided eye of the examiner; and yet, withal, the vision in these cases may be, for the letter test, normal both for distance and in accommodation? If, again, the primary reflex response to light be abolished, why does that fact not leave the sympathetic free to act with a resulting double dilatation of the pupil? Or, again, why are there abolished at the same time with the primary light reflex all of those sympathetic extracerebral reflexes which result in dilatation of the pupil? The answer to these questions requires first an understanding of what takes place in the case of a normal pupil contracting and dilating as the impinging light rays vary in intensity. Increase the intensity of the light and the pupil contracts—lessen it and the pupil dilates. How is this brought about? Lessen the intensity of the light impulses and the stimulation transferred to the sphincter fibers in the ciliary ganglia is lessened, but how does this result in dilatation of the pupil—a thing which is accomplished by the dilatator muscle and this

has a sympathetic innervation? Again, shut out the light from one eye and the pupil of the other eye dilates—even though the previous illumination of the second eye remains unaltered. Thus we see that lessening the amount of light falling upon the back of one or both eyes results in dilatation of both pupils.

The influences which cause contraction of the pupil—that is, increase the action of the motor fibers, result in restraint being put upon the action of the sympathetic fibers, and we again get a glimpse of the necessity for the two sets of fibers in the optic nerves, and of the necessity for two sets of terminal differentiations of these nerve fibers—the rods and the cones. Let us now destroy the sensori-motor and sensori-sympathetic connections in the ciliary ganglion and we do away with the primary light reflex. We do away at the same time with all sympathetic action upon the dilatator iridis, which comes through and is modified by the ciliary ganglion—in other words, we have done away with the *raison d'être* of the ciliary ganglion, and the only impulses which reach the sphincter iridis are those which pass along the motor fibers from the third-nerve nuclei. Contraction of the pupil is, therefore, a necessary result; in other words, the result is an immobile pupil so far as the ciliary ganglia can influence its dilating or contracting fibers—*i.e.*, the motor impulses from the third-nerve nuclei, originating from all sources other than the ciliary ganglion itself, are still active and the result is the contracted pupil of reflex iridoplegia. For I take it for granted that the process at work within the ciliary ganglion confines its action often at first and for a long time thereafter—often for many years—to the sensory connecting elements within the ganglion. The sympathetic fibers are a part of the great sympathetic tract from the subthalamic sympathetic center to the sphincter. A part of this tract passes to the ciliary ganglion and a part *via* the long ciliary nerves to the sphincter. The latter part influences the dilatator iridis in the great and necessary central function above referred to, of keeping the pupils in accommodation equal in size. The effect of the sympathetic impulses that pass from the extracerebral and extraspinal ganglia to the ciliary ganglia is lost, owing to destruction of the sympathetic connection within the ciliary ganglia; while the

action of the sympathetic from the long ciliary nerves is retained as manifested in the fact that both pupils are kept of the same size in the act of convergence. I have given what I believe to be the seat of the lesion which causes the reflex iridoplegia found in the Argyll–Robertson pupil and I have tried to explain my reasons for my belief. Whether time will prove these views to be correct will be seen.

Why, in some cases of the Argyll–Robertson phenomenon, is the size of the pupil larger than in others? Several factors contribute to make this so. In some eyes the width and thickness of the iris are different from others and, as a rule, the larger and thinner the iris the larger the pupil; in other words, developmental differences come into play. Or, again, the toxin at work in the ciliary ganglion cannot always be expected to be as exact in the limits of its action in some ganglia as in others and this could conceivably, and probably does actually, determine some of the differences under question. What have we to say of the hemianopic pupillary reflex—both when the lesion is in the optic tract anterior to the point where the fibers to the third-nerve nuclei are given off and when the lesion is posterior to this point? I shall first answer the former half of this question. When the lesion is in the optic tract anterior to the point where the fibers to the corpora quadrigemina and the subthalamic ganglia are given off and is of sufficient extent to produce a typical hemianopsia, then from the blind halves of both retinæ there can be awakened by light impulses neither element of the light reflex. This is the usually accepted statement of the case. I think it is only partially correct. I believe the following will be found to be true. Immediately following the lesion which causes the hemianopsia, light falling upon the blind halves of the retinæ will fail to arouse the consensual reflex, for no retinal-born impulses can reach the quadrigeminal region. How about the autonomic, the primary light reflex? The "blind half" of each retina will respond to light till degeneration of the retinal cells takes place. The primary light reflex cannot then be aroused by the impact of light on the blind halves of the retinæ because, owing to the degeneration of the retinal fibers to these areas, there is no activity in the pigment cells in response to light rays. Make these retinal cells irresponsive to light

impulses and the primary light reflex will cease. When the lesion is posterior to the point where the fibers to the third nerve and to the subthalamic ganglion are given off, we find that light impulses to the retina awaken both the primary and the consensual light reflexes. This is as it should be, for in such lesions degeneration of the optic fibers anterior to the corpora quadrigemina and the subthalamic ganglion do not take place and the retinal cells remain responsive to light impulses. I have not recently had an opportunity to examine the picture presented in the Wernicke reflex and I take the text-book statement for its details. The following case, however, is in this connection most interesting and instructive.

On Dec. 25, 1916, Mr. L. aged 26, was struck by a locomotive engine and among other injuries was a blow over the left eye. Thirty-six hours later he was examined by Dr. W. W. Gill of Richmond who noted that when light was thrown into the patient's left eye there was a momentary weak contraction followed immediately by a wide dilatation. The sight of the eye was much impaired. Owing to the condition of the patient no attempt was made to determine exactly how much. The right eye was normal and the consensual reflexes normal. On Jan. 1, 1917, examination of the left eye showed the following: entire abolition of vision save for the outer part of the nasal portion of the retina. The nerve head was snowy white except for a triangular section in the inner half of the disk. When light was thrown upon the macula region there was no pupillary response. When light was thrown upon the outer half of the retina there was no pupillary response. The symptom of the "rebounding" pupil had disappeared—but when the eye was turned toward the nose as far as possible and the light thrown upon the retina the pupil contracted. This response was distinct. The interpretation of these symptoms I take to be as follows: Thirty-six hours after the injury to the nerve the retinal elements had not degenerated and the primary light reflex, as described above, was possible. The impulses imparted by the light to the retinal cells arouse impulses in the pigment cells and these were carried to the ciliary region where they were noted by the sensitive nerve ends and thence transmitted to the ciliary ganglion, whence efferent impulses reached the sphincter pupillæ and produced a momentary contraction—*i.e.*, the primary light reflex—but no impulses went from the retina along the optic nerve to the third-nerve nuclei and so no central efferent contracting impulses were

aroused to maintain and to increase the pupillary contraction. The dilating impulses were then unopposed and the pupil rebounded, *i. e.*, dilated to their action. Naturally the consensual reaction remained unimpaired so far as keeping the two pupils of the same size was concerned. One week later the retinal cells had, for the central and outer portions and part of the inner portions of the retina, degenerated. Now no impulses passed from the retinal cells to the pigment cells and the primary light impulse was abolished and with it the rebounding pupil and all pupillary light responses from the affected areas. The primary light reflex was still present for the portion of the retina which had escaped—*i.e.*, the extreme inner nasal fibers. Of course, the details of this response could not be accurately studied owing to the position of the eye while the tests were made.

Material for microscopic purposes in the early cases of Argyll–Robertson pupil is not easy to obtain. Owing to the long duration of the disease and the changes which finally take place in the ciliary ganglia, the seat of the specific process, the material when available is useless for our purposes. That the specific toxins should select both ciliary ganglia and so produce bilateral pupillary changes is no more to be wondered at than other syphilitic manifestations. That the specific toxins should select definite portions of the ciliary ganglion to the neglect of others is no more to be wondered at than that they should select the ganglia themselves. Of course, until we know accurately the minute anatomy of the ciliary ganglion we can only speculate as to the exact location of the primary lesion and its subsequent degenerations. For analogy I would refer you to the pictures of the selective action of the syphilitic poison to be seen in the posterior spinal ganglia in locomotor ataxia, the primary lesion in which lies in the posterior spinal ganglia, in the sensory neurons thereof. And we have, in the abolition of the patellar reflex, a condition analogous to what we see in the Argyll–Robertson pupil. To work out the details of this analogy here is unnecessary. Whether syphilis furnishes the only toxins capable of exerting selective action upon definite portions of the ciliary ganglia, I do not know. Those who claim that the Argyll–Robertson pupil is found only in this disease should bear in mind the French saying: "Le mot, toujours, n'existe pas dans le médecine." That an overwhelmingly large proportion of all cases are of syphilitic

origin is of course beyond question. I omit here all detailed explanation of the minor variations in type of the Argyll–Robertson pupil. Bearing in mind the pathology I have tried to make clear, these variations are easily understood.

I would however briefly ask attention to one or two of them. *As time goes on it is possible for the toxins, directly and indirectly, to affect the motor portion of the ciliary ganglion, and this results in relative dilatation of the pupil and the abolition of pupillary response in convergence—i. e., in loss of all pupillary responses.*

These changes may affect one ganglion more than the other, or sooner than the other, and inequality of the pupils results. This observation, to my mind, if interpreted aright, does away, once and for all, with the possibility of the seat of the lesion in the Argyll–Robertson pupil being situated in any other place than in the ciliary ganglia. The conclusion would seem clear and absolute. We must not forget that our picture may be complicated by co-existing specific involvement of some other portion of our pupillary nervous tract. Where this is the case, our typical pupillary picture suffers change. Of these changes, the easiest to detect is seen where there is involvement of one or both of the third-nerve nuclei or the fibers passing therefrom. Again there may co-exist lesions in the cervical sympathetic and once more our typical picture suffers change. I have often noticed in cases of the Argyll–Robertson phenomenon of long standing, those in which secondary pupillary inequality has set in, that there co-exists evidence of retinal degeneration. *E.g.*

Mr. L., aged 52. Duration of constitutional trouble fifteen years. V. O. D. fingers at 3 feet. O. S., fingers at 2 feet. Both pupils of Argyll–Robertson type. Left pupil slightly larger than right—both contracted but not *ad maximum*. There is no peripheral contraction of the fields for light, although there seem to be one or more relatively large absolute scotomata. This is judged to be the fact rather from the patient's expression as the position of the light is varied, than from his answers, which are uncertain. The retina shows irregular local pigment disturbances and a general lack of transparency, more marked in some places than others. The retinal arteries and veins are much contracted. The nerve head showed advanced atrophy.

My interpretation of this picture is as follows: The beginning secondary dilatation of the pupils, I think, shows advanced changes in the ciliary ganglia with beginning destruction of the transganglionic third-nerve fibers. The opaque, abnormally colored retinæ with their visible aggregations of retinal pigment I take to show that the sensory fibers from the ciliary ganglia to the ciliary pigment cells have atrophied and that a secondary degeneration of the retinal pigment cells and thence a consecutive degeneration of the retinal cells has set in, producing the retinal vascular picture and the white nerve heads. While I do not insist absolutely on the correctness of this interpretation, the more cases of the late types of the Argyll–Robertson pupil I see the more probable it seems. I do not mean to say that there do not occur other types of optic atrophy with the Argyll–Robertson pupil. There do occur such types, but they have essential visible differences from the one I am trying to describe. Comments on this interesting chapter of the Argyll–Robertson pupil are omitted here, except to say that my observations seem to show that when, as explained above, the contraction of the Argyll–Robertson pupil gives way to relative dilatation this is accompanied by relative loss of vision. In conclusion, I believe that the complete type of the Argyll–Robertson pupil cannot be produced by a lesion anywhere save in the ciliary ganglion, and even there cannot be produced save by the selective action of a toxin or germ, which up to the present has been shown to be the result only of syphilitic infection.

SUMMARY.

The primary response of the pupil to light is a reflex belonging to the autonomic system. Its path is fully described above. That the rods represent the terminals of the fibers to the subthalamic ganglion and the cones the terminals to the anterior quadrigeminal and external geniculate bodies is a suggestion that came from consideration, first, of the necessity for two sets of optic fiber terminals, and, second, of the existence of the consensual reflex. Whether the suggestion be purely fanciful or not, it opens the way for a number of interesting questions and seemingly answers some of them. The

Argyll-Robertson pupil is the result of the abolition of the autonomic reflex of the ciliary ganglia. The suggestion that the varied picture of the Argyll-Robertson pupil may be explained by the extent of the lesions within the ciliary ganglia and the subsequent degenerations therein is, I think, borne out by analogous pathology elsewhere. The contention that certain features of the optico-retinal pictures occurring along with the Argyll-Robertson pupil in its late stages are due to degeneration of the sensory fibers from the ciliary ganglia to the ciliary region and thence, by the pigment layer, to the retina will, I think, prove to be correct.

THE EXCISION OF THE TARSUS IN TRACHOMA. COMMENTS THEREON.[1]

By GEORGE FRANCIS SUKER, M.D., Chicago.

(With 7 line cuts in the text.)

IN this country the excision of the tarsus, together with the overlying conjunctiva to within 1 or 1½*mm* of the margin of the lid, and the covering of this area by conjunctiva drawn from the upper retrotarsal cul-de-sac is not practiced as much as abroad.

For this there are several reasons: principal among which is the dread that an entropion may ensue; another, when the tarsal space is not covered by conjunctiva, it is filled in with granulation tissue which in time contracts, eventually producing an entropion; finally, some fear that after an excision of the tarsus the regular contour of the lid edge becomes more or less distorted. Then too, that a veritable lagophthalmos may result from the excision of the tarsus, because of the undue retraction, has deterred some from availing themselves of this useful and beneficial operation. That a possible ptosis may also result from undue damage to the levator has been a drawback to the operation as ordinarily performed.

No doubt, unless a strict surgical technic is observed and the proper judgment exercised in determining how far one should remain away from the free border of the lid, and how much conjunctiva is to be sacrificed, and how far to carry the dissection up into the retrotarsal fold, some one or more of these supposed untoward conditions may possibly arise.

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[1] From the ophthalmic service in Cook County Hospital, Chicago, Ill.

Read at the Memphis meeting of American Academy of Ophthalmology and Oto-Laryngology, December 11, 12, 13, 1916.

Another factor which deters some operators from employing this operation is their indecision in the proper selection of cases. This latter difficulty is not a hard one to surmount.

Upon general principles it may be stated that the operation is indicated in:

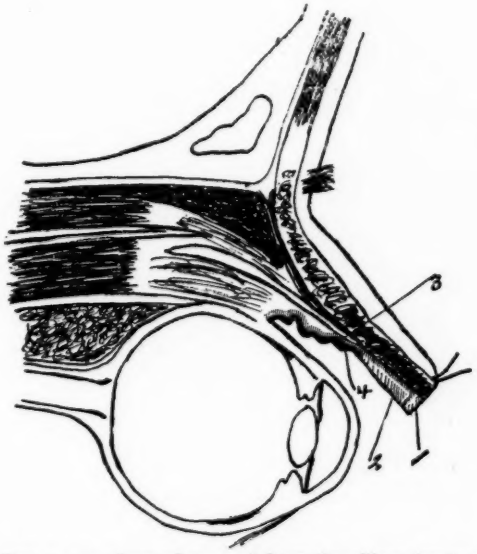

FIG. 1.—1. 1½*mm* of tarsus left at the ciliary border. The cut is made perpendicular and parallel to lid edge. 2. Portion of tarsus to be excised. 3. Line of incision and its direction from ciliary edge to cul-de-sac. 4. Retrotarsal conjunctival fold.

(*a*) Any case of trachoma which does not yield to the usual line of treatment, no matter how vigorously or conscientiously the same has been executed;

(*b*) In cases of so-called "hot box"—*i.e.*, in cases wherein the tarsus is extensively cicatrized, uneven, and more or less studded with islands of vascularized pinhead nodes, which constantly irritate the cornea and cause a persistent but attenuated pannus. The tarsus itself may not be greatly incurvated;

(*c*) In cases in which the tarsus is comparatively smooth, yet greatly incurvated through cicatrization, thus exerting undue pressure upon the cornea, causing ulcers in the same and vascularization at the limbus;

(*d*) In cases of a velvety, infiltrated tarsal conjunctiva with more or less extensive pannus, such as are often seen in the old exuberant chronic trachoma, which apparently yielded

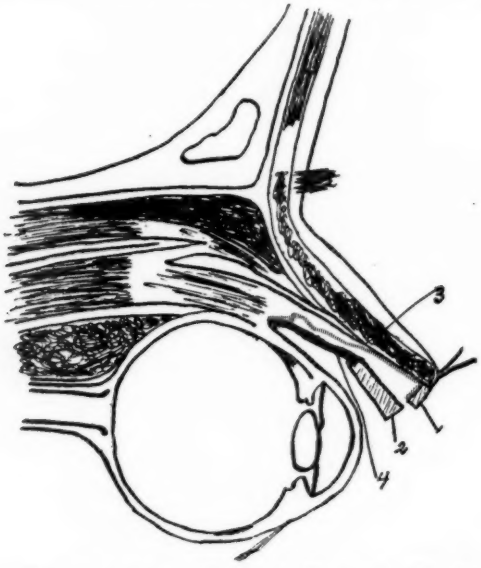

FIG. 2.—1, 2, 3, 4. Same as in Figure 1. After the incision is made.

nicely in the acute stages, but in which the too heroic measures have set up this proliferative and velvety conjunctiva which ultimately cicatrizes extensively and incurvates the tarsus;

(*e*) In cases in which the upper half of the cornea is frequently subjected to ulcers after an apparent disappearance of the trachoma;

(*f*) In cases in which the apparent cure resulted in producing an entropion with its usual train of symptoms;

(*g*) In cases in which the pannus does not satisfactorily subside, though the tarsus and retrotarsal folds are

seemingly in a fair state of aggravation, as it were, to either cause or maintain said pannus;

(*h*) In cases in which the tarsus was grattaged and left distorted and studded with connective-tissue pin-point nodules.

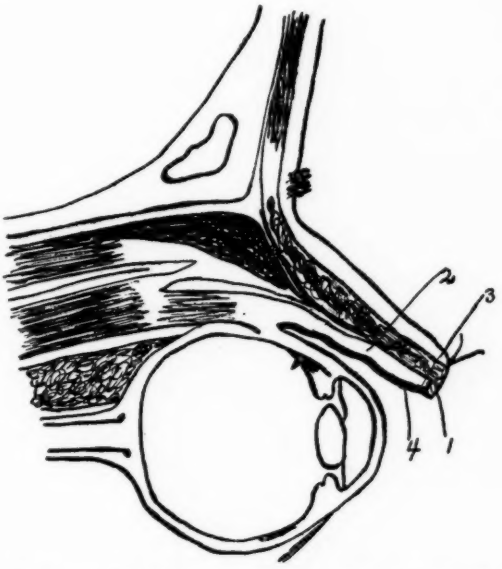

FIG. 3.—1. Strip of tarsus at ciliary border. 2. So-called dead space between skin (lid) and retrotarsal conjunctiva. 3. Conjunctiva brought to lid margin (strip of tarsus). 4. Retrotarsal conjunctiva brought down after dissection as far as possible.

On the other hand, it may be stated, in broad terms, that the excision of the tarsus is not indicated when the cornea is not extensively involved, either as an acute affair or a chronic one.

The presence of corneal ulcers is no contraindication for delaying excision of the tarsus; on the contrary, it is an indication for the immediate performance of it.

As long as the cornea remains free from so-called gravity ulcerations at the pupillary margin of the pannus and that

this pannus is apparently quiescent, the tarsus need not be excised. Should, however, this pannus give the appearance of a marginal infiltration or thickening at its pupillary border, then the tarsus should be excised. In these cases the tarsus may and often is in a state of essential cicatrization, with

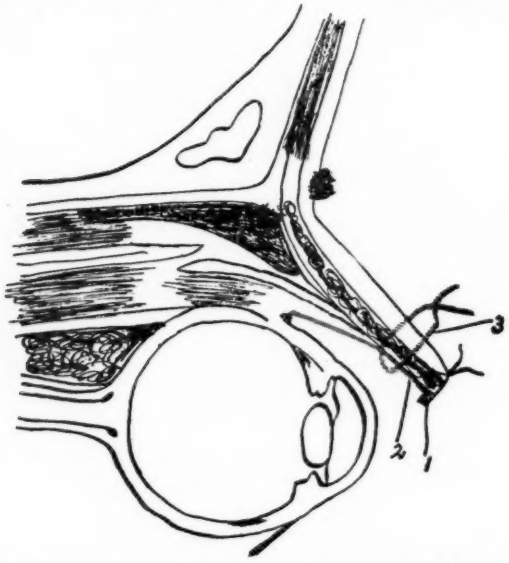

FIG. 4.—1, 2, same as Fig. 3. 3. Suture passes from conjunctival surface of lid to skin surface and tied over small pledgets of cotton. Two of these usually necessary (placed at inner and outer third of lid).

minute connective-tissue ridges or rugæ, and yet appears relatively smooth, still causing sufficient friction to the cornea.

Given a case suitable for excision, in order to obtain the best surgical and therapeutic results, the following suggestions are to be observed:

1. Always leave 1–1½*mm* strip of the tarsus at the ciliary margin of the lid. This strip of tarsus is seldom if ever involved in the trachomatous process (Fig. 2).

2. Begin the excision at the ciliary border of the lid—not at the retrotarsal border (Figs. 1 and 2).

3. Do not ever attempt to save the conjunctiva directly overlying the tarsus.

4. Do not excise more than just the tarsus.

5. Dissect the conjunctiva far enough, throughout the entire width of the lid into the retrotarsal fold, taking care not to have much more than just sufficient conjunctiva to comfortably cover, without much tension, the area formerly occupied by the tarsus (Fig. 3).

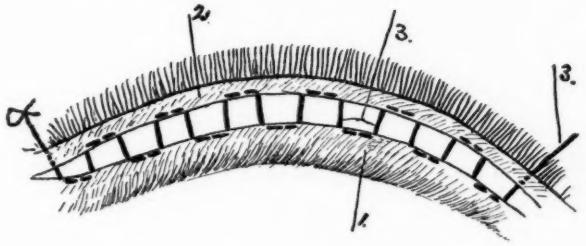

FIG. 5.—1. Conjunctiva (Tarsus excised). 2. Tarsal strip, cut surface. 3. Course of suture.

Pass the suture from the skin surface, at either inner or outer canthus (where it is fixed or tied on the skin surface), through the tarsal strip and then into the substance of the conjunctiva, back into the substance of the tarsal strip (from the cut edge) and back again into the conjunctiva. Continue in this manner until the whole margin of the conjunctival flap is neatly approximated to the cut edge of the tarsal strip. The last stitch passes out through on to the skin—tension is made so as to take up any slack. It is now suitably anchored at this point.

6. Do not excise tarsus until the retrotarsal fold has been dissected as mentioned. This avoids undue manipulation with forceps of the edge of the conjunctiva to be attached to the ciliary edge of the tarsus (not at all an unimportant step in the operation).

7. Secure a good approximation of this edge of the retrotarsal conjunctiva to the 1 or 1½*mm* tarsal strip left at the ciliary margin of the lid.

8. Employ a mattress suture for securing this requisite coaptation just mentioned (subconjunctival and subtarsal, Fig. 5).

9. Obliterate the "dead space" necessarily produced between the lid and the fold by the stretching of this retrotarsal conjunctiva, by two properly placed sutures at a point corresponding to the previous retrotarsal border of the tarsus (Fig. 4). This will transform the retrotarsal conjunctiva and lid into one, and with sutures as mentioned at the former retrotarsal edge the lid will not retract nor will entropion take place, as the anatomical insertion of the levator is thus properly maintained and ptosis does not supervene. Traction upward is exerted from this newly established line of cicatrization.

FIG. 6.—Tarsal Clamp. Within the fenestrum the tarsus is dissected from the ciliary border backward towards the retrotarsal fold. Before releasing this clamp, the T forceps are applied to the dissected tarsus and then the tarsal clamp is slipped over the T forceps for the continuation of the dissection.

10. Do not attempt to do this operation under a local anæsthetic; under local anæsthesia you cannot get the relaxation of tissue as under a general anæsthesia. Then, too, the infiltration caused by the local anæsthetic injection is very annoying.

After the operation, apply a goodly quantity of sterile vaseline or White's ointment and then apply a rather snug bandage. As soon as the patient recovers sufficiently from the anæsthetic, apply an ice-bag directly over the bandage, which must not be too voluminous. Keep this ice-bag on for several alternating hours for at least twenty-four or forty-eight hours. By doing this you considerably lessen the surgical reaction.

The sutures which are needed to establish the new insertion for the levator and obliterate the dead space (see Fig. 4) can be removed on the second or third day after operation.

The mattress suture (Fig. 5) along the lid edge can be safely removed on the third or fourth, but not later than on the fourth day subsequent to the operation.

The lid clamp and forceps which have been found useful in this operation are simple and readily applied (Illustrations 6 and 7).

FIG. 7.—Two of these T forceps are very convenient for the excising of the tarsus. One is applied to the portion of the tarsus after same is dissected down from the ciliary border to the retrotarsal edge in the fenestrum of the tarsal forceps—when the tarsal forceps are unlocked and slipped over the T forceps. The second T forceps is now applied to the lid. Then an assistant can hold both and the operator dissect farther into retrotarsal fold without the use of other forceps. Thus the T forceps answer a triple purpose—control hemorrhage, avoid undue manipulations with other forceps, and make the dissection easy in so far that the whole width of lid and tarsus is within their grasp. When the dissection is carried sufficiently far, then the tarsus is excised and both forceps released.

The accompanying illustrations perhaps convey a better idea of the technic than a verbal description.

It is advisable, after the removal of all sutures, to use moderate astringents on the upper as well as the lower conjunctiva. For, even after the removal of the tarsus, the retrotarsal fold, as is usual, is more or less subacutely inflamed and needs attention. This is a point often lost sight of by those who think the removal of the tarsus is a panacea. Within a very short time, under the judicious employment of astringents, massage, and the like, the conjunctiva becomes very smooth and remains so.

It is not the lower conjunctiva in trachoma that is the

bête noire, but the upper. And, as a rule, the trachomatous process is seldom as pernicious as in the upper lid.

In my service at the Cook County Hospital, my Assistants, Drs. Coppes and Carr, have performed many such an excision with practically universal satisfactory cures—*i.e.*, brought the process to an end and aided materially in minimizing the disastrous corneal opacities and pannus and doing away with the ever recurring faceted ulcers in the cornea. In each instance, the annoying little comebacks, as they might be called, were done away with. The expression "annoying little comebacks" in old pseudo-active trachoma is more or less apropos and is readily understood by all as to what it means.

In conclusion, it may be stated that perhaps there are several new steps in performing this operation which differ from the methods usually described. And, since we have adopted these modifications in my service, the excision of the tarsus for conditions as above detailed has proven the only satisfactory operation.

DIPLOBACILLI IN CORNEAL ULCERS.

By HUNTER W. SCARLETT, M.D., Philadelphia.

IN June, 1896, Morax (1) described the diplobacillus causing subacute conjunctivitis. Three months later this finding was confirmed by Axenfeld. In 1899 Petit (2) isolated a diplobacillus morphologically resembling that of Morax, but differing from it in some cultural characteristics. Chaillous, in 1905, found in the scraping of a corneal ulcer a diplobacillus similar in every respect to the Morax bacillus except that it was Gram positive.

While routinely studying the bacteriological factor in corneal ulcers, the writer isolated, in 1915, a diplobacillus differing from those already mentioned, in some of its cultural characteristics and in its humoral reactions, though resembling them in morphological structure.

There are many who contend that the various diplobacilli are one and the same organism, yet the studies of Petit and Chaine (3) and our own findings seem to clearly separate them as later demonstrated.

In the above-mentioned routine examination of central ulcers of the cornea, where the diplobacillus was present, the latter proved almost invariably to be that of Petit, while marginal ulcers revealed the presence of the Morax bacillus.

One case, described below, was particularly interesting because of the presence of an organism which, although similar in form to the diplobacilli of Morax and Petit, was different from these in cultural growth.

It was found in a central ulcer of the cornea, in a woman aged 67. The patient complained of severe orbital and periorbital pain, with diminution of vision in the right eye,

which had existed for ten days. Several days previous to this attack, she had been troubled with profuse lacrimation. When first seen her vision amounted to light perception only.

Previous History.—Several years ago, patient was treated for constricted lacrimal ducts. About two years ago, she had a small corneal ulcer of the right eye, following a slight trauma.

Examination showed a large well defined ulcer about *2mm* in diameter, which colored with fluorescin, located in the center of the right cornea. Eyeball was markedly injected and tender. Tension was slightly elevated. Anterior chamber showed a hypopyon of *1½mm.* Iris was injected. No synechia present. Lacrimal passages were permeable.

Bacteriological examination of the ulcer revealed the presence of a few Gram-negative diplobacilli of the same size and shape as the diplobacillus of Morax.

Treatment consisted of lavage, atropine, touching ulcer with 2½% solution of zinc sulphate. Condition grew worse and the hypopyon increased. Ulcer was then cauterized with the galvanocautery, and cornea perforated, allowing the pus to escape. Pain then became less severe, the ulcer commenced to heal, and the injection of the eyeball disappeared. At the end of a month, there remained a grayish-white leucoma in the center of the cornea, which permitted the patient to see the movements of the hand only.

Bacteriological Examination.—A scraping from the base and sides of the ulcer revealed a few Gram-negative diplobacilli, but no other organisms.

A tube of ascitic agar was inoculated and in twenty-four hours showed a growth of several small, round colonies about *1½mm* in diameter, with transparent edges and opaque centers. Smears of the colonies showed a Gram-negative diplobacillus. From this culture inoculations were made on slants of ordinary agar, coagulated serum, potato and carrot and the various sugars, also in stab gelatin, bouillon and milk, and on petri plates of agar. Growth occurred on all except potato, carrot, and in milk. There was no fermentation of the sugars.

The point of interest in differentiating this organism from that of Morax and Petit, lies in the fact that it grew in gelatin without causing any liquefaction, and on coagulated serum without any erosion or liquefaction. Being Gram negative,

it may be distinguished from the "Bacillus Duplex of Chaillous."

Animal inoculations into the anterior chamber, into the layers of the cornea, on the scraped surface of the cornea, into the conjunctival sac, and under the skin, produced no lesions, showing that this organism was not pathogenic for laboratory animals.

Because of its ability to grow in stab gelatin and on coagulated serum without producing any liquefaction or erosion, this diplobacillus has been named "Bacillus Duplex Non-Liquefaciens," to distinguish it from the "Bacillus Duplex" of Morax, and the "Bacillus Duplex Liquefaciens" of Petit.

A glance at the following chart will readily show the differential characters of the four diplobacilli mentioned, as regards their cultural and staining properties.

DIFFERENTIAL FEATURES IN CULTURE AND IN STAINING.

	Bacillus Duplex	*Bacillus Duplex Liquefaciens*	*Bacillus Duplex Non-Liquefaciens*	*Bacillus Duplex of Chaillous*
Coagulated Serum	Growth, erosion, and liquefaction	Growth and liquefaction	Growth, no liquefaction	Growth slight, no liquefaction
Gelatin	No growth	Growth and liquefaction	Growth, no liquefaction	No growth
Ordinary Agar	No growth	Growth	Growth	No growth
Ordinary Temperature	No growth	Growth	Growth	
Gram Stain	Negative	Negative	Negative	Positive

To further distinguish the "Bacillus Duplex Non-Liquefaciens" from the other diplobacilli, experiments were made to determine the humoral reactions of the serums of animals inoculated with "Bacillus Duplex," "Bacillus Duplex Liquefaciens," and "Bacillus Duplex Non-Liquefaciens." The same method was used as that employed by Chaine in his work of differentiating the diplobacillus of Morax and that of Petit. He found that the serum of a rabbit inoculated with the Morax bacillus would agglutinate that organism and cause a precipitation, but would do neither in the case of the Petit bacillus. Also the serum of a rabbit treated with the bacillus of Petit

was specific, and would neither agglutinate the bacillus of Morax, nor cause a precipitation.

Our results confirmed his findings and we further demonstrated that the "Bacillus Duplex Non-Liquefaciens" produced a serum specific for that organism and having no effect on the other two.

The serums were prepared by inoculating three rabbits, one for each organism. A twenty-four-hour culture, mixed with physiological salt solution in the dilutions of 1:4, 1:3, 1:2, 1:1, was employed; 1*cc* of the first three dilutions and 2*cc* of the last dilution being injected into the vein of an ear. Injections were made at intervals from three to six days.

Two weeks after the last injection, the blood was collected directly from the carotid artery into sterile test tubes, and placed in the ice-box for three days. The clot was then removed and the serum collected in other sterile tubes, ready for the experiments of precipitation and agglutination.

CONCLUSIONS.

Because the "Bacillus Duplex Non-Liquefaciens" will grow in gelatin and produce no liquefaction and will cause no erosion or liquefaction on coagulated serum, and when inoculated into rabbits will produce a serum containing specific agglutinins and precipitins, the writer feels that this organism is distinct from that of Morax and Petit.

A résumé of twenty cases of corneal ulcer in which the diplobacillus was found, revealed the presence of the Morax bacillus in six, that of Petit in thirteen, and the "Bacillus Duplex Non-Liquefaciens" in one. The types of ulcer encountered were divided into central and marginal. In the marginal ulcers, only the bacillus of Morax was present, while the central ulcers showed the bacillus of Petit in all but two cases, the Morax bacillus being present in one and the "Bacillus Duplex Non-Liquefaciens" in the other.

It is interesting to note that the marginal ulcers healed quickly and with little or no impairment of the corneal tissue, and were never accompanied by hypopyon.

On the contrary, the central ulcers were more severe, were accompanied by hypopyon in all but three cases, healed with

difficulty, and in every case there remained a corneal scar which interfered with the vision of the eye, frequently reducing it to perception of the hand alone.

In central ulcers where the diplobacillus was discovered, an examination of the conjunctival secretion failed to reveal the presence of any organism save possibly a few cocci or Massué bacilli, but in the marginal type the diplobacillus of Morax was generally found in the conjunctival sac.

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TENDO-MUSCLE LENGTHENING AND DESCRIPTION OF SPECIAL FORCEPS.

By HOLBROOK LOWELL, M.D.,

Assistant Surgeon, Mass. Char. Eye and Ear Infirmary; Ex-Assistant Surgeon, Boston City Hospital.

(*With one illustration in the text.*)

A FORM of partial tenotomy was proposed by Verhoeff in 1903 (1) which he called "A Graduated Plastic Tenotomy." "Each edge of the tendon is divided at the insertion and again 5*mm* from the insertion. Halfway between these positions the central fibers are divided." In this operation *no fiber is left uncut.* This method marked a step in advance of previously described partial tenotomies (2), but it was very difficult to perform.

In 1907 Todd described an operation (3) entitled "A Method of Performing Tenotomy which Enables the Operator to Limit the Effect as Required." The operation consists in making only two cuts in the extra-ocular muscle tendon, one on one side and one on the other side. This method was another step forward as it was more practical and more easily performed then Verhoeff's.

It fell to Harmon in January, 1913 (4), to enlarge upon Todd's method by increasing the number of cuts in the tendon from two to three, two on one side and one on the opposite side, thus allowing greater lengthening and better control.

In October, 1914 (5), the writer reported results of cases done by this three-cut method.

In December, 1914 (6), Todd reported case results by the three-cut method and again in June, 1916 (7), refers to the three-cut method.

In each of the above communications, including the writer's, the operation has been specified as one performed on a tendon. This the writer now believes to be inaccurate because the tendon of the greatest length is that of the external rectus muscle, approximately 8.8*mm*, and the shortest that of the internal rectus muscle, approximately 3.7*mm*. From the above data one may see how difficult it is to confine this operation to such a small field with any hope of accurate technic.

The writer attributes much of the uniformity of his good results to the fact that, although one cut may have been in tendon, at least two cuts were in muscle tissue. This weakened the muscle without any appreciable limitation of motion and gave greater primary and therefore greater final lengthening.

For the above reasons this should be named "An Extra-Ocular Tendo-Muscle Lengthening Operation" as this title is clearly descriptive and not misleading. The name "Partial Myotomy" has been used, suggesting a partial cutting of a muscle, but this does not hint at the purpose for so doing.

The writer's operation was done without any special instrument, strabismus hooks and scissors being used. Later he procured a special forceps devised by Harmon. These are not easily manipulated and do not give a clear field for action. It is difficult to perform this operation without special holding forceps, because the severed muscle ends stretch apart immediately after being cut, and one is not sure of the spaces between the cuts, or just how far in the muscle has been divided.

The writer feels that many operators have refrained from performing this most useful operation in heterophoria and in conjunction with shortening operations for strabismus because of the aforesaid difficulties. He has therefore devised a double-jawed fixation forceps (see illustration), the use of which overcomes these obstacles. The muscle is held securely between the jaws and the space of 4*mm* between them is

ample in which to operate and the relations are not lost, thereby allowing for greater accuracy. Where the jaws begin at the handle they bend at an angle of 135° and are toothed to prevent any slipping. They are 11*mm* long and will grasp a muscle much wider than is likely to be met. There is ample room to allow for the middle cut from behind with angle scissors. The sliding fixation catch is sure and easily manipulated.

This instrument was made for me by Codman & Shurtleff, of Boston.

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DACRYOCYSTORHINOSTOMY.

By ALFRED WIENER, M.D., New York.

IT is my firm belief, that the operation of dacryocystorhinostomy will bring relief to patients suffering from epiphora and the complications produced by obstruction of the lacrimal apparatus. Accumulating evidence from the reports of such operators as West, Polvak, Salus, Mosher, and Schirmer, is ample proof that we as ophthalmologists should encourage its performance. The principle of this operation is to establish a direct communication between the sac and the nose. Hirschberg tells us that this idea is an ancient one, having been suggested and practiced by Galen, Celsus, and Paul V. Aegina many, many years ago. Others followed with their various methods, amongst them Langier and Foltz, who suggested, instead of making an opening into the nose, to enter the antrum. In 1904 Toti suggested an operation which on account of its simplicity appealed to many. Under general anæsthesia he exposed the sac and the lacrimal fossa. From this fossa he resected an oval piece of bone extending from the postlacrimal crest downwards as far as the lacrimal duct, and upwards as far as the highest point of the sac. He then removed the inner wall of the sac, and the nasal mucous membrane corresponding to the opening in the bone. Light tampons were made throughout the wound and nose, and the skin over this sutured. At times a complete resection of the middle turbinated bone or its anterior end was practiced. In spite of the fact that this method offered much, many surgeons still clung to conservative measures. Hess advised permanent sounds; Koster permanent drainage by means of a silk suture moistened with silver: Tartuferi a slitting of the upper canaliculus and internal palpebral ligament, curettage

of the sac, and its treatment with sounds and antiseptic solutions.

The intranasal operation, with sacrifice of tissue and probing, recommended by Polvak, Killian, and Passow, but without much practical experience, did not appeal to many. A great disadvantage was its long-continued after-treatment and control. The placing of foreign substances such as decalcified bone tubes (Guital), after opening the sac and curettage, was not successful. The sac prothesis (Zimmerman method) was condemned both by Kuhnt and Schirmer as an unsuitable method for replacing the functions of the sac. As these methods proved one after the other disappointing, we drifted to the approved radical cutaneous method of extirpation of the sac, as taught by Kuhnt, Czermak, and Axenfeld. While this offered a cure for the diseased sac, it possessed some disadvantages. The natural passageway for the tears, etc., had been destroyed. The epiphora which remained, while only slightly evident at times, at other times was excessive, and could not be controlled even by removal of the accessory tear gland. Besides the above mentioned procedures practiced for the purpose of curing chronic suppuration of the sac, chronic recurring suppuration of the sac with or without fistula, obstruction or incurable obliteration of the lacrimal apparatus below the canaliculi, I admit there are many others. It is practically impossible for me in this short paper to review them all. My purpose is to emphasize the fact that the operation of dacryocystorhinostomy finds its indication in those cases in which epiphora and its complications are due to an obstruction within or below the sac. Recently, more than ever, there has been an endeavor to replace the painful probing of the lacrimal passages for epiphora by this operation. It is evident that probing if persisted in may result in a permanent cure. Experience has taught most of us, however, that few patients are willing to undergo the long-continued and painful discomfort attending such treatment.

At the present time there is a choice of two routes in the operation of dacryocystorhinostomy. The external, of which the Toti operation is the representative one, and the other, the intranasal, as practiced by Polvak, West, Mosher, and Yankauer. It appears to me that the operative measure, which is simple

and offers and accomplishes a satisfactory result, should be the one to appeal most strongly to the surgeon. Of the two routes the intranasal is the more difficult one to perform. It requires especial technical skill limited to those who are skillful in nasal surgery. I can claim, from my own experience, that it accomplishes not one bit more than the external operation, if the latter is performed in a skillful manner. My object in this paper is to point out the advantage of the Toti operation, but performed with some refinements in the technic. This to my mind makes it an ideal operation. One has at all times a clear view of the operative field. This is most important. It happens not infrequently that an ethmoidal cell intervenes between the sac and the nasal cavity. When this occurs, it is dealt with much more satisfactorily than when such a cell is encountered in the intranasal operation. There is no danger of entering the orbital cavity, or antrum, or intracranial cavity, as a possible calamity, mentioned by intranasal operators. The modified Toti operation as practiced by myself is as follows: It is preferably done under local anæsthesia, but can be performed under general anæsthesia. I had occasion to perform it once under general anæsthesia on a boy ten years of age. Under local anæsthesia I proceed in the following manner: About *2cc* of the following sterile solution is used: novocain two grams, normal salt solution one-half strength one hundred grams, and ten drops of adrenalin chloride one to one-thousand. The first puncture with the hypodermic syringe is made at the inner orbital margin at the height of the root of the nose. The needle is passed horizontally backwards close to the bone to a depth of about *2cm.* Inject a few drops of the solution. Then as one withdraws the needle inject at short intervals a few more drops. In this way in the majority of cases the ethmoidal nerve is blocked. Anæsthesia is obtained of the inner portion of the upper and lower eyelids, the tear sac, together with the superior and anterior part of the nasal mucosa. A few drops are injected along the line of incision and deep under the periosteum in the lacrimal fossa. A piece of cotton, saturated with ten per cent. cocaine and one to one-thousand adrenalin, is placed intranasally just below and in the front of the anterior end of the middle turbinated bone. With the field prepared in this

way the operation is practically painless. The sac is exposed in the usual manner, separating at once the nasal or inner wall from the lacrimal fossa. If found closely adherent, it is better to make an incision into the sac from above downwards just where the anterior wall is reflected on to the bone. This of course opens the sac at once. With a curette, or by dissection, clean off this layer of mucoperiosteum from the lacrimal fossa until you get a clear and clean picture of the bone cavity. This brings into view the posterior lip of the maxillary process and the portion of the lacrimal bone lying anterior to its posterior crest. A trephine of about 4*mm* in diameter is placed as low down in the lacrimal fossa as possible, and a small button of bone removed. With a small conchotome the bone is bitten away until an opening about 6 to 10*mm* is made. A sharp-pointed probe passed through the mucous membrane at the bottom of this opening should be visible on intranasal inspection. If possible a flap is made with the base down, quadrilateral in shape, and representing the bone opening. If not, cut away the mucous membrane entirely so that the opening is free and permits the remaining portion of the sac to adapt itself thereto. If one is able to obtain a flap, reflect it back into the sac and then place the packing. I make use of selvage-edge iodoform gauze, one-half inch in width and saturated with vaseline. This is pushed into the nose in liberal quantity. The upper end is held fast by a strand of Pagenstecher suture passed through the lower canaliculus. This suture can be threaded through the lower canaliculus by means of a thin lacrimal probe containing an eyelet. To the lower end of the packing, a second suture is fastened, and then the packing is carefully pushed and drawn back into the cavity of the sac. The lower and upper sutures are folded and attached to the forehead and cheek with adhesive. The upper suture should be about five inches in length, in order to permit a renewal of the packing after the first dressing is made. This is accomplished by simply pulling on the lower suture until the dressing is removed and the upper suture appears at the anterior nares.[1] We now go back to the sac and with chromic acid gut stitch its cut anterior edge to the periosteum of the anterior edge of the opening.

[1] A method similar to one suggested by Mosher.

Then the skin is sutured over the sac and a plain dressing applied. The intranasal dressing is changed on the third day unless there is some indication for doing so earlier. About three dressings are made. Gentle probing is kept up for about one month, two or three times a week. The intranasal opening is treated through the nose. It is necessary to watch for granulation tissue and remove it by gentle cauterization. I have performed this operation twelve times and have every reason to feel that it has given me most satisfactory results. The first case was operated upon two years ago and reported to me only recently for observation. The patient is entirely free from her epiphora and chronic dacryocystitis. She has had no discomfort of any kind since the operation. The fourth case, operated upon one year ago, needs occasional probing after recurrent attacks of rhinitis. All the others have shown no discomfort whatsoever since their respective operations. In none of the cases have I been able to express any secretion from the sac. I propose to publish in detail all these cases after a larger number have been operated upon. Suffice it to say that among the above cases were three with chronic dacryocystitis of several years' duration, two with ectasia of the sac in which probing had been unsuccessful, and two of recurrent phlegmonous inflammation, one with fistula.

After the Toti operation recurrences have been reported. I believe that they will also be reported at times from the above modification. However, in these cases the cause will be found, as Schirmer has explained it, to be due to the complete disturbance of the drainage mechanism. Patients, in whom constant probing and too extensive slitting of the canaliculi have taken place, will therefore not be the proper cases for selection. When the canaliculi have been extensively slit, there is the discomfort of having air and nasal secretion blown back into the conjunctival sac. In one of my cases, where this occurred, the patient went through an attack of acute rhinitis without the slightest evidence of any infection of the sac or conjunctiva. By less vigorous blowing of the nose this fault was held in obeyance.

In all patients selected for this operation the nose should be carefully examined. In chronic progressive disease of this organ the operation finds a contraindication. Where there is

marked deviation of the septum, or where the middle turbinated bone is laterally deflected, these defects should be corrected before the operation. Excessive polypoid formation should be thoroughly dealt with. A resection of the anterior end of the middle turbinated bone was necessary in one of my cases. This should be done whenever it interferes with the proper placing or making of the intranasal opening.

One naturally feels puzzled just why in such a complicated physiological mechanism, the drainage of the tears and secretion should be so easily accomplished through artificial means. Schirmer has given us the probable correct explanation. He believes that the truly physiological mechanism is replaced by the self-contractility of the canaliculi and with the help of aspiration from tne nose.

The claim made by the opponents of the external operation, that the scar made is unsightly and disfiguring, is not borne out by facts. I wish most emphatically to contradict this. If the operation is properly performed, I claim that the scar after six weeks is scarcely perceptible.

I believe that Mosher is correct, when he says that the operation of dacryocystorhinostomy, like other operations devised to accomplish the same end, faces the danger of a gradual narrowing and final cicatricial closure of the opening. The remedy however should be a simple one. It can easily be reopened by slitting intranasally, or probing through the canaliculus externally, with a minimum amount of discomfort.

I believe this operation as described, or with still better modifications, to be the one of choice. It is technically not difficult and in the majority of cases will bring about the desired result.

FIBROLYSIN IN THE TREATMENT OF OCULAR DISEASES.[1]

By ROBERT SCOTT LAMB, M.D., Washington, D. C.

BEFORE proceeding with my story let me express my thanks to you for honoring me with your invitation to address you; my endeavor shall be to merit your approval.

While tarrying in Colorado Springs, with others who had attended a meeting of the "Colorado Ophthalmological Congress" two years ago, it was my good fortune to find companionship with Drs. Parke Lewis of Buffalo, and Wm. Luedde of St. Louis; and opportunity for discussion of our special pet treatments of different ocular conditions. And really you know there seems to be nothing more valuable in connection with these meetings of ours than the communication held between us—sort of confidential statements regarding our methods of handling various diseases; our hopes for special benefit to be derived from some particular drug or practice; our ambitions for our friends, ourselves, medical education, and other things equally important to us individually and collectively.

In the course of such events it fell out that Dr. Luedde had been using fibrolysin in the treatment of vernal catarrh and from experience believed it to be a specific cure. In fact he was so enthusiastic that I believed him and, all evidence to the contrary notwithstanding, I made trial of the treatment described by him.

My first attempt was to corral six chronic recurrent typical and well marked cases, well known to have resisted all usual methods, put them in the hospital under control, and

[1] Read before the New England Ophthalmological Society, Feb. 13. 1917.

use each night at bedtime one drop of fibrolysin in each eye. Imagine my astonishment to find at the end of ten days all were well. They were discharged from the hospital and requested to return to the out-patient department for observation once a week. There was no more vernal catarrh among them that season. Such a happening could do but one thing—namely, require a search of the literature for information regarding the drug and a study, first, of the reasons for the discontinuance of its use; second, the conclusions arrived at by those who had reported upon it; and last, why they had given out the impression to the majority of us that it was of indifferent worth in our armamentarium.

All this entailed the expenditure of time and effort, but nevertheless led to some experimental work and therapeutic application; the results of which I have the pleasure to bring before you to-night.

Fibrolysin is a trade name for a sterilized solution of thiosinamine and sodium salicylate—15% of the double salt in water, put up in ampoules—2.3*cc* each. Thiosinamine, or allyl-sulphocarbamide, is a mixture of volatile oil of mustard and an alcoholic solution of ammonium 10% in water—sufficient glycerine added to keep it in solution.

Fibrolysin was first introduced to the medical profession in 1905 by Mendel of Essen, who presented, among other evidences of its efficacy, its effectual cure of Dupuytren's contraction, which as all know was considered wellnigh incurable. Following this were a few short papers in Italian, French, German, American, and English literature, and an occasional report over the following years; but for the most part the value of the drug has been overlooked; to say the least the mental attitude has been indifferent.

Its application in eye diseases has been limited to chronic or cicatricial conditions. Unless perhaps Suker meant by plastic iritis and exudative choroiditis to include more recent changes. The allied substance thiosinamine was employed by Brandenberg in corneal opacity following keratitis scrofula, keratitis purulenta, keratitis trachomatosa, chronic uveitis, and chronic retrobulbar neuritis. I merely mention this in passing for I don't want to be understood to take to myself undue credit for observations or results herewith recorded.

As I have stated, its use seems to have been limited to chronic or cicatricial forms of disease, to soften the scar and so help in its absorption, or render it sufficiently thin and pliable to enable sounds or lacrimal probes to be passed, or to produce easier motion of an eyelid or other ocular appendage. The theory of the effect of the fibrolysin on cicatricial tissue was as follows: swelling, the individual fibres lose their sharpness of outline, the nuclei are parted, the tissues become spongy and more pliable. Grossmann thought it due to serous infiltration softening the hard, old inflammatory tissues, in a manner akin to the hyperæmia method of Bier. The absorption effected was increased by repeated injections, and the administration was by intravenous, intramuscular, or subcutaneous injections.

In trying to digest these and other facts, while ruminating upon them, it occurred to me that if injections of fibrolysin could produce such favorable result upon hard old cicatricial tissues, how much more could they do towards resorption of recent forming or healing tissues, to prevent scarring, disadvantageous alike from either a cosmetic or an utilitarian standpoint. As a result of the experiments which followed and the cures and otherwise beneficial effects of practical therapeutic administration, I have to report the following: Before and 12 to 20 hours after injections, blood counts were made for the purpose of ascertaining especially the effect on the white cells. A lymphocytosis following injections, occurred regularly, indicating at least one possible explanation of the modus operandi of fibrolysin when introduced into the system. Blood pressure taken before and after injections showed a fall in pressure, apparently directly dependent upon the injection, ranging from 15 to 25*mm* of mercury, directly proportionate to the height of the systolic pressure, so that when the systolic pressure was lower than normal there occurred in some cases an almost toxic effect, in others the low systolic pressure was brought up to normal. This I have attributed to a probable stimulation, by the drug, of the entire defense mechanism of the internal secretory system, thereby restoring the balance to approximate or relative normal. Paradoxic as it may seem, it was not the only strange result obtained during the series of observations. Except for the rare cases of depression following temporarily an unusually large injection, or a usual dose

in an unusually sensitive or debilitated person, the feeling of improvement in wellbeing was usual, immediate, and steadily ascending as the treatment proceeded. Other observations of a general character made in connection wih a study of the effect of the drug upon the general system are passed over without comment as having no bearing on our present paper.

Turning to the practical application of these findings as recorded we see a never-ending field of usefulness only limited by a penetrative, analytic imagination and an inadequate supply of the drug. The second statement is easily understood; the first may need a word of explanation. There are those among us who try a new remedy upon numerous ailments without due regard to the fundamental principles of the practice of medicine, who have imagination, yet lead themselves astray, and are as apt to lead others by the very brilliancy and plausibility of their suggestions. Others there are who always take the well established, well trodden way, who, though safer, can but progress as the snail does, slowly. There may be other kinds but for our purpose there are those by whom progress is made, and they have a good foundation by inheritance and education, a good penetrative imagination, well controlled by analysis of their problems, and a self-analysis keen enough to prevent prejudice over-persuading them. These are our ideal men of the profession and our purpose and endeavor is to some day become one of their number by recording the findings of our experiments and our treatments of ocular disease in such a manner as to merit respect and be believed. This should be the goal for each of us for therein lies lasting progress for us all.

The cases about to be discussed are but a very few of the many treated with fibrolysin during the years just passed. Some had the drug instilled in the cul-de-sac; others were injected subcutaneously with it, and some got both; this last during that period only when there was apparently an unlimited supply.

One of the early cases was a child of 6 years of age who had been struck in the right eye with the branch of a thorn bush, tearing through the cornea and sclera, including that part overlying the ciliary body on both nasal and temporal sides of the limbus; the eye was collapsed and looked worth-

less. Remembering Jones of Cumberland and his cyanide of mercury injection, I proceeded to follow his directions, thereby making antiseptic the eyeball and surroundings. After some ten days' treatment along lines well established and authoritative for the absorption of blood from the anterior chamber—the iris was still not apparent because of a complete filling of the anterior chamber with blood or perhaps blood clot, or both,—I gave an injection of fibrolysin 1*cc* and was astounded the next day to be able to see the iris clearly when the eye bandage was removed, for the blood was entirely absorbed. Other injections followed from time to time and the eye became quiet—although all the hemorrhage into the vitreous was not absorbed when he left the hospital. He was allowed to use 5% dionin and given prescription for syr. ferri iodidi to be taken internally. Three months later he was in town and I had to examine very carefully to ascertain which eye had been injured; the eyes appeared normal and there was neither vertical nor horizontal deviation.

Another early case was that of a man 45 years old upon whom one of my clinical assistants did an enucleation of the right eye because of iridocyclitis. Following operation there was an immense hæmatoma formed in the orbital tissues, which parted the eyelids and extended for 2½ inches in front of the orbital margins. After two weeks of treatment with ergot, iron, and calcium lactate, iodide and local applications, blood was still oozing from the center of the mass. My attention was called to it and Kocher's Coagulen was injected; the bleeding stopped but the mass remained for a week—when a dose of 2.3*cc* fibrolysin was given and the mass began to shrink. After eight injections four days apart the mass had shrunken sufficiently to allow the lids to move over it as over an ordinary eyeball. He was kept under observation for a while and then allowed to go to his home.

Another relatively early case was that of a physician—age 38—who had previously had two slight intraocular hemorrhages and both had been absorbed without any impairment of vision to be observed. When seen, the right eye was able to distinguish light doubtfully—there was a complete filling of the vitreous chamber with blood from hemorrhage. Usual absorptive treatment including iodides was begun, but iodism called a halt. I described to him what my results had been with fibrolysin in numerous cases and he became desirous of trying it. Therefore injections were begun—at first at regular intervals; then, as he improved and went on with his professional duties incident to a large country practice, the intervals became irregular and longer;

nevertheless at the end of 13 months he could read without a glass with this eye, and though there were numerous floaters, large and small, one could see the details of the fundus.

A ball player came to me suffering with a double optic neuroretinitis—worse O. S.—etiology syphilis—confirmed by blood and spinal Noguchi test and cell count. Marked exudate and much swelling. Vision: fingers, 2 ft. Treatment, modern, approved. First visit February, 1916; began to play ball again in April, 1916. Was able to go through season, and to-day has $\frac{9}{4}$ vision; without glass reads J. 1. Fibrolysin injections were given during that period prior to the definite diagnosis determined by Noguchi test and at intervals between the intravenous and intraspinous injections of salvarsan and salvarsanized serum which were given and checked by count from time to time during the summer. The striking result which may be attributed to the fibrolysin is that the disks are nearly alike now, even under the Gullstrand. This, I think, could hardly have happened otherwise in a case of virulent syphilitic infection of the nervous system with a start of two months' manifest symptoms, preceding the first visit to my office.

There are so many kinds of diseases in which this substance can be used to advantage I hardly know which way to turn first to tell you about them. Suppose we take the group of ulcerative keratitis, recurrent and almost chronic. To illustrate:

F. J., recurrent, corneal ulcers of both eyes, alternating; history of 9 years' duration; cured in 10 months, with almost no scarring remaining. (Remains cured after 2 years this last fall.)

Another type due to tubercular infection. Positive to O. T. $^1/_{10,000}$ of *mg*, with focal reaction, which cleared under fibrolysin even after the picture was transformed from keratitis ulcerativa to keratitis tuberculosa with marked exudate. Of these one was a young woman:

R. C., 23 years of age, who had ulcers for four years; reacted strongly to tuberculin—even having tubercular follicles of conjunctiva.

Another young woman, R., age 26; similar condition for five years. Reacted so strongly that the tubercular follicles are still manifest after eight months; but the ulcers

remain cured and general health with concomitant weight increase is much improved.

Young girl, L. B., age 13 years; positive reaction to O. T. $^1/_{10,000}$ *mg*—keratoiridocyclitis, and is now in the hospital; plastic exudate so dense that iris could not be seen; distortion of eyeball and beginning staphyloma which is now under control; eyeball normal shape and iris now clearly visible after six weeks' treatment, even though the injections could not be regularly given owing to lack of medicine.

A girl, 10 years of age, typical case of dished face—Hutchinsonian teeth, etc., yet the keratitis—plastic exudate dense and obscuring pupils—is arranged as distinct star-shaped plaques—so numerous as to seem to be a homogeneous mass until examined under the corneal microscope when true character is visible. Treated previously for two years for interstitial keratitis with mercury and iodides without improvement; when seen by me intercurrent tubercular infection was suspected; given tuberculin $^1/_{10,000}$ *mg*—was positive; two months' treatment with fibrolysin; entire iris evident and only scattered discrete plaques visible on cornea except with microscope, which shows connecting bands. Whole cornea clearing well and general health improving simultaneously—only one injection a week—dose 1*cc*.

Another case of peculiar interest is that of M., who had been treated in Paris, London, New York, since last summer. He was attached to the American Embassy in Paris when a unilateral keratitis began; after various treatments he fell into the hands of one of my colleagues in Washington by whom he was treated for six weeks for interstitial keratitis. As improvement was either slow or uncertain I had the pleasure of seeing the patient. Wassermann +++. The right eye had an exudative keratitis circumscribed—possibly a gumma, or perhaps a tubercular exudative mass; it occupied an area the shape of a quarter moon with the hollow up, the ends rounded, and the lower edge below the center of the pupil ⅛ inch. It did not extend to the limbus in any direction, but was connected with the vascular structures of the corneoscleral margins by a number of vessels hardly dense enough to be described by the term vascularizing keratitis. The corneal microscope showed the composition of the mass to be an aggregation of numerous gray fibers, swollen and apparently infiltrated with accumulation of lymphocytes. I suggested fibrolysin and a dose of 2.3*cc* was given—apparently an overdose, because of the marked reaction which followed; the next day the entire iris angle was filled with a ring of lymphocytes and the patient could make out objects about the room for the first time in four

months. Some days later the sight continued improving and the lymphocytes were all absorbed. The dose was not repeated by the attending surgeon as I desired, but injections of mercury in the buttocks were begun instead. The surgeon, though acknowledging the benefit of the tremendous reaction, was inclined to await a period of non-improvement before trying the drug again. Moral: it is wise to have control of the cases on which a new treatment is being tried. For example, I should have started an acute case with 1*cc* or less of the drug, and worked up gradually to 2.3*cc*.[1]

In another class of cases fibrolysin is most useful—those exudative retinitis cases, forerunners of hemorrhagic retinitis; and even of hemorrhagic retinitis itself, associated usually with a cardiorenal complex. I have found it an excellent treatment. Many such cases, even including those with marked vitreous hemorrhage, were satisfactorily treated—by which I mean cured except for floaters,—and some entirely restored to relative normality, considering age and general condition. Iridic adhesions which have resisted all usual forms of treatment frequently have broken and become absorbed following its use. Hyphæma has invariably responded, and cortical substance resisting ordinary methods for absorption has been rapidly influenced and absorption accelerated.

I have used fibrolysin in iritis, uveitis, choroiditis in selected cases with satisfaction, and have found it particularly efficacious in preventing ugly and hard fibrous scars following plastic operations upon the eye and its appendages.

Far be it from me to leave with you the impression that fibrolysin can be used with impunity or that it is a panacea for all ocular ills. There are disappointments, disadvantages, and possible dangers in its use. For instance, Mr. and Mrs. H., both getting on in years, for they have been married 49 years, had angiosclerotic changes in lenses and fundi, and whereas in each of them the first dose was very satisfactory from every viewpoint, there was after some one of the subsequent injections a very marked depression, with nausea, vomiting for several hours, and weakness for several days. Other similar cases have appeared from time to time until I became con-

[1] *Subsequent history.*

vinced that it was no coincidence but a direct result of the administration of the drug; since then the initial dose has been proportioned to the body weight and the apparent resistability of the person. Therefore similar reactions have not lately occurred.

The burning sensation at the site of the injection is severe for several minutes, then it disappears. The lump which occasionally forms may go away in time, but can be assisted to absorb by mercury friction applied over the surrounding area. The solution decomposes quickly when exposed to air and should be used only when fresh. The injection is often followed by black and blue marks, and on two occasions when injecting into the back over the trapezius muscle, in women whose waists were not pulled low enough for me to make sure of my location, the injection became subcuticular and there was a slough which was never septic but took a long time to heal and dermatize.

What can be said that you have not already heard? I believe you have been realizing that what I have said relates directly to the results of an immense amount of work on numerous individuals and of many, many observations of a critical character which have been made.

Because of limited time the cases herein briefly reported may seem to be such as might have been treated otherwise as satisfactorily and brought to end results quite as good as were obtained with fibrolysin. I cannot deny the possibility, nor have I a prejudicial desire so to do; yet would I crave your gentlest handling of my discourse until such time as you may satisfy yourselves that I have warrant for the statements made or that perchance I may have erred in giving too much credit to fibrolysin.

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A CASE OF CONGENITAL ENOPHTHALMOS IN CONNECTION WITH COMPLETE ABSENCE OF ABDUCTION AND RETRACTION OF THE EYEBALL IN ADDUCTION.

By ANTON LUTZ, M.D., Habana, Cuba.

(With six illustrations on Text-Plates III and IV.)

THERE are only very few publications on *Congenital Defects of the Motility of the Eye*, in connection with retraction of the globe. Birch-Hirschfeld collected out of the whole literature up to 1907, only fifty cases, which are tabulated in his monograph, *The Diseases of the Orbit* (Graefe-Saemisch). These cases are characterized by the following points:

a.—In all cases abduction is abolished either completely or almost completely, and in adduction (more or less possible) there occurs a retraction of the eyeball with the production of enophthalmos. From the fact that retraction in case of adduction was always found together with deficiency of abduction, there was good ground to conclude that the retraction and the deficiency of the motility were produced by the same cause.

b.—Convergence is mostly defective and the palpebral fissure is narrowed through action of the orbicularis, which may be seen in the active elevation of the lower eyelid.

c.—The anomaly has always been found to be unilateral, except in one case, and is seen mostly in females (30 females to 20 men), and three times as often on the left side as on the right one (35 left side to 10 right).

The enophthalmos could be noticed in the primary position in only ⅕ of the cases, *i. e.* in 13 cases, and there exist only

three observations (Best and Bergmeister) in which the enophthalmos was not increased in adduction. The highest degree of retraction was found in the case of Alling, 10 millimeters.

Anatomically there was found, instead of the abducens, a fibrous band, which explains the deficiency of abduction.

It has been attempted to explain the retraction of the eyeball by abnormal shortness of the rectus internus, and by its insertion farther back than normal, so that the muscle could not turn the eyeball inward, but had to pull it back. More plausible is the view of Hirschfeld, who considers the internus of normal length and normally inserted, but that it had to pull the eyeball backward as soon as the fibrous band (rectus externus) got tense.

Finally, Treacher Collins calls attention to the fact that the fibrous bands which go from Tenon's capsule to the periosteum of the orbit prevent the muscles from pulling back the eyeball. These bands being defective or inserted too far back, retraction must follow.

The enophthalmos was explained by the action of the muscles, because it was transformed into exophthalmos by abduction in five cases (Wolff, Knapp, Schapringer), or by congenital hemorrhages in the orbit, with following atrophy of the orbital tissue (Bergmeister). Only in one case (Dupuys-Dutemps) were there found fibrous bands fixing the eyeball.

Thus far, no alteration has been found in the bones of the orbit.

The extreme rareness of these cases induces me to publish the following:

R. I., 26 years old, mechanic, tells us that his mother died from hepatic cirrhosis, his father from asthma; his sisters being healthy. In his family there is no history of ocular anomalies, of consanguinity, etc. He says that he was born with two similar and healthy eyes; that in the first month of his life he had a fever, after which some change was noticed in his right eye. He never suffered from any disease nor recollects any traumatism in the face. At a distance it can be noticed that the right eye lies deeper than the left one.

ILLUSTRATING DR. LUTZ'S ARTICLE ON "CONGENITAL ENOPHTHALMOS."

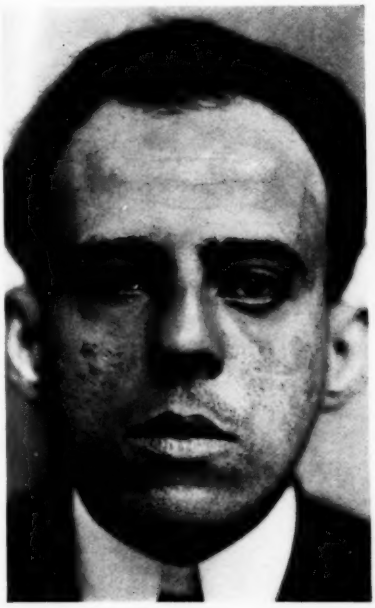

FIG. 1.
Enophthalmos.

FIG. 2.
Looking upward; Enophthalmos better seen.

FIG. 3.
Looking to the right. Movement of eyeball is stopped just in the center line.

FIG. 4.
Deficiency of the right rectus internus in convergence.

FIG. 5.
Looking to the left. Retraction of the right eyeball, the palpebral fissure getting at the same time smaller.

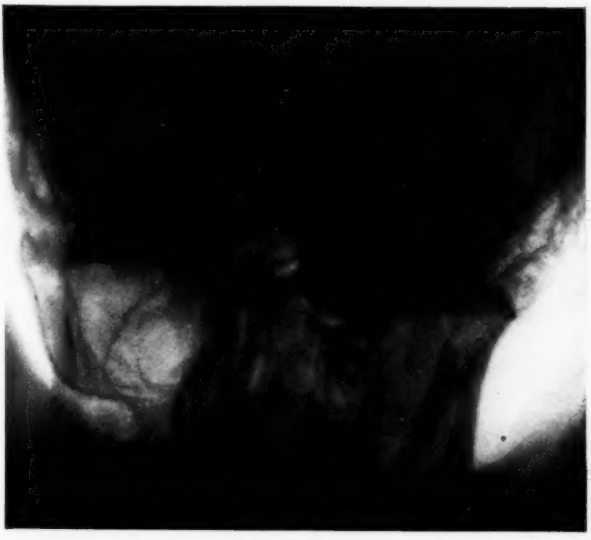

FIG. 6.
X-plate with face on the plate showing opacity in right orbit.

Javal R. E. = 1.0 against the rule.
L. E. = 0.25 with the rule.
Vision R. E. = $^{3}/_{60}$. Small central scotoma for colors.

$$\text{L. E.} = \frac{4.5}{4.5} + 1.5 = \frac{4.5}{3.0}$$

Media transparent, fundi normal, field of vision normal for colors and moving objects.

Pupillary reaction in both eyes; normal to light (direct as well as consensual), to accommodation, to convergence, and to irritation of the trigeminus; no difference in the size of the pupils.

No anomaly of the sympathetic of the eye or of the face. No pronounced assymmetry of the face.

There exists no diplopia, spontaneous or by means of a red glass.

No abnormal movement can be found in the lids or the lips.

In the primary position no strabismus can be observed, but there is a very marked enophthalmos (*vide* Fig. 1), which is still easier to be seen when the patient looks upwards (Fig. 2). Binocular movement is normal upwards and downwards. Monocular movement of the left eye is normal in all directions; on the right eye there exists complete absence of the function of the externus (*vide* Fig. 3), the movement of the eyeball stopping just in the center line; no secondary deviation is to be found. Monocular movement up and downwards quite normal. Adduction exists, but a certain deficiency in the action of the rectus internus can undoubtedly be seen in convergence (*vide* Fig. 4); when the patient looks to the left side, a marked retraction of the bulbus takes place (*vide* Fig. 5); the eyeball is pulled back, the slit between the eyelids becomes smaller by elevation of the lower eyelid and drooping of the upper one (contraction of the orbicularis), the eyelids showing at the same time a certain quivering, as if they should have lost their support.

Palpebral closure is good on both sides, the facial and the other cranial nerves showing no anomaly.

The radiogram, which I owe to the kindness of Prof. Francisco Dominguez-Roldan, clearly shows that the apex of the orbit is less transparent on the right side, indicating increased ossification in the bones in this situation. As such an ossification diminishes normally the capacity of the orbit the adipose tissue of the orbit must atrophy. According to the anamnesis the patient suffered from fever early in life which possibly led to an inflammation at the apex of the orbit] (periostitis), and caused paralysis of the abducens and retardation of the development of the orbit.

The above mentioned case is in accordance with the majority of the other publications:

a.—In the complete absence of the function of the abducens, without secondary deviation, and without diplopia.

b.—In a certain weakness of adduction in convergence.

c.—In the retraction of the eyeball when looking towards the healthy side.

It differs from the majority of the other publications:

a.—By the very marked enophthalmos in the primary position.

b.—By the radiographic findings.

A CASE OF OPTIC HALLUCINATIONS WITH HEMIANOPSIA.[1]

By OTTO SCHIRMER, M. D., New York.

The patient, whom I have the honor to present to the Section, is a lady of 45 years of age. She says that her general health is perfect. A careful examination by an internist could not be made, because she objected. Her blood-pressure was slightly increased, 165. The superficial arteries are not rigid. The impression she makes is perfectly normal, both mentally and bodily. The configuration of her face reminds one to some extent of acromegaly, but no other signs are available. I saw the lady first two years ago, when she consulted me for reading-glasses. The eyes at that time were perfectly normal.

The patient consulted me again three weeks ago with the following history: On January 16th she awoke with a mild headache and dizziness. She took a book and tried to read. All letters were perfectly distinct and she had no difficulty to read a line, but it was a hard task to find the next line, and she was successful only after a few futile attempts. The diagnosis of left-sided homonymous hemianopsia, for which this complaint is characteristic, was corroborated by her observation that she was inclined to run against objects at her left side.

All these symptoms mentioned disappeared in a few days, and when I saw the patient a week later she was convinced that her eyes were practically normal. The examination corroborated this statement. Vision and objective findings were normal. The fields showed a very slight contraction (10–20°) on both left sides; there was no color hemianopsia; but objects in both halves of the field, seen under the same angle and under the same illumination, showed a marked difference: the object in the right half appeared much brighter.

So far the case does not show any extraordinary symp-

[1] Read before the Ophthalmological Section of the N. Y. Academy.

toms. It was a left homonymous hemianopsia, which might have been due, either to a slight hemorrhage in the posterior part of the right internal capsule, or to a thrombosis in the right occipital region. A peculiar feature is the rapid retrogression of the hemianopsia. This seems to point to a location not within, but in the region adjacent to, the areas mentioned. Hemianopsia as a distant symptom is not a rare occurrence. I have seen several cases of hemiplegia with passing hemianopsia and I believe we would find such cases much oftener, if we could examine the hemiplegic patient shortly after the attack. I have seen furthermore several cases of lasting hemianopsia, which appeared suddenly with moderate brain symptoms and were complicated by a passing disturbance of motion, especially of the finer movements of the hand on the same side. Here the abortive hemiplegic phenomena are the distant symptoms; they are due to a slight lesion of the pyramidal tracts, which are located in the internal capsule, in the immediate neighborhood of Gratiolet's radiating fibres which were the seat of the hemorrhage.

A hemorrhage or thrombosis always produces œdema and therefore pressure in the neighborhood. Furthermore chemical changes develop and exert their harmful effect on the nerve fibres or ganglion cells. So I feel entitled to diagnose in my case a focus not within, but in the immediate neighborhood of, Gratiolet's fibres or of the psycho-optic center. The latter explanation seems more likely to me, because no symptoms from the pyramidal tracts were present.

This case furthermore presents a symptom of unusual interest, not only on account of its rarity, but because it permits of a more exact focal diagnosis. The patient states that on the same day and also on the following days she saw a very good-looking boy with dark hair and black eyes seated on her left at a distance of 1–2 yards. As soon as she tried to focus on the boy, he disappeared towards the left side. A few times she saw a girl in the same place. At other times a crowd of men appeared to her. All were moving with considerable speed towards her left side and disappeared there. These phenomena were most marked and sharply outlined on the first day. They lost quickly in distinctness during the first week. When I saw the patient it required some effort on her part to reproduce these pictures, and the recollection mingled with what she really saw. At no time was she in doubt that she did not see real objects; at no time was she frightened by the same. She has never had any mental disturbance; she is not even of the nervous type.

There is no doubt that we have to deal here with optic hallucinations. We distinguish, as you know, between hallucinations and illusions. Illusions are wrong interpretations of a retinal image. The patient sees a towel on the wall and thinks it is a woman; or he sees the shadow of a vitreous opacity and thinks it is a mouse. Hallucinations arise without any stimulation of the peripheral visual organ.

Hallucinations with homonymous hemianopsia are a very rare occurrence. Uhthoff, who has searched the whole literature, has found 30 cases only. Hallucinations in the cases with post-mortem autopsies were always due to a focus in the occipital lobe, to a direct irritation at the ganglion cells of the optic center. Only the case of De Schweinitz is different. Here the post-mortem revealed the presence of a gummatous affection of the optic tract, as the cause of the hemianopsia and the hallucinations.

The hallucinatory images always appear in the blind halves. This is what one should expect *a priori*, because the focus paralyzes the majority of the ganglion-cells, thus producing hemianopsia. At the same time it causes an irritation of the remaining ganglion cells on the same side and produces the hallucinations. There are two cases of Henschen on record where the hallucinations were in the seeing halves. It is an interesting fact that in these cases the post-mortem revealed a thrombotic destruction of one optic center and at the same time slight morbid changes in the other optic lobe. Furthermore, Henschen, based on his post-mortem findings, states that hallucinations are more apt to occur when the focus is not in the optic center, but in its neighborhood, mostly at the lateral side of the occipital lobe.

In this case as in all cases on record the hallucinatory pictures were complete; they contained both sides of the object; they were not hemianopic. They therefore corresponded not to the perceptions, but to the conceptions, of the patient.

I do not believe that this is a sufficient reason for us to locate the lesions beyond the psycho-optic center into a higher center, as we have to, for instance, in order to explain the unilateral hysterical blindness. The anatomical findings are directly contrary to such hypothesis.

Adding the hallucinations to the other facts of the history, I feel entitled to say that the lesion in this case was located in the right occipital lobe, in the neighborhood of the psycho-optic center. It was in all probability a thrombosis.

REPORT OF A CASE OF TOXIC CHOROIDITIS SECONDARY TO A CHRONIC ALVEOLAR ABSCESS, SUBSEQUENTLY TREATED WITH VACCINE THERAPY.

By E. HARRISON ROWE, M.D., Baltimore, Md.

Ophthalmic Surgeon, Baltimore Eye, Ear, Nose, and Throat Charity Hospital; Assistant Ophthalmologist, Johns Hopkins Hospital Dispensary

(*With two illustrations on Text-Plate V.*)

On Dec. 4, 1916, Miss E. B., 25 years old, consulted me because of failing sight in the right eye. The vision was O. D. fingers at 10 feet; O. S. $^{20}/_{13}$. Interrogated at this point, patient remembers always seeing better with the right eye while at school. She has never had any pain or ocular discomfort.

Examination of right eye: Lids normal; bulbar excursions normal; conjunctiva bulbi normal; slight, if any, pericorneal injection. Cornea normal in appearance. Sphincter pupillæ actively responded to light. Iris, under examination with binocular loupe, seemed devoid of inflammatory changes. Numerous deposits were seen on Descemet's membrane, varying from 1 to 2*mm* in diameter, which faded away into a diffuse cloudiness at the lower quadrant. Tension was normal on palpation. Homatropin was instilled and the pupil dilated evenly to 8*mm*. Ophthalmoscopic examination showed dense vitreous cloudiness with many floating opacities. The eye ground was not seen. On inspection, the left eye appeared to be normal. Ophthalmoscopic examination showed an area of choroidal degeneration, with exposed sclera and pigment changes, a little less in size than the optic disk, and regular in contour. This patch extended about two disk diameters into the macular region. The optic nerve was pale. No other changes were noted.

Diagnosis: Right eye: Diffuse choroiditis with involvement of the ciliary tract. Left eye: From the uniform appearance of the choroidal changes they are possibly congenital. The only history worthy of comment at this time was that she had recently been under the care of her dentist. As neither the patient nor her parents could see the wisdom of a Wassermann test, she was dismissed with a pair of dark glasses and local treatment of atropin and dionin and told to return at a later date. After further consultation, it was finally decided that a radiographic examination of her mouth be made. I have the pleasure of adding a note from Dr. Arthur Lankford, who was kind enough to supply the accompanying photographs.

"*Report on mouth examination:* Radiograph made Dec. 12, operated on Dec. 14, 1916. When patient reported for mouth examination she had a history of choroiditis of several weeks' standing, eyesight badly impaired, considerable loss in weight, and no appetite. Mouth diagnosis showed all teeth vital and in good condition with the exception of the two upper central incisors and radiographs showed these teeth to have been devitalized and the roots imperfectly filled. A large necrotic area involving the apical region of both of these teeth was plainly shown. On questioning the patient in regard to these teeth it was found that she had had trouble with them about a year prior to this time, when the teeth were treated and the roots filled. Shortly afterward a fistula appeared; this was touched up from time to time with some medicament, the patient did not know what was used. The fistula finally closed and the patient was told she was cured. Shortly after the closing of this fistula the patient noticed trouble with the right eye which steadily grew worse. The two teeth involved were removed. One contained at its apex a tough fibrous sack the size of a large pea, the other showed complete disintegration of the apex by necrosis. Exploration of the bone above these teeth showed badly necrotic condition extending back under the nares. All necrotic bone was removed, leaving the anterior half of the floor of the nose protected by soft tissue. After the operation the patient experienced considerable nervousness which subsided at the end of two days. One week after the operation the patient reported much improvement in her sight and appetite returning. Wound was healing nicely; at the end of two weeks patient reported continued improvement of eyesight and the wound filling in nicely. Have not had the opportunity of seeing patient since."

Dec. 23, 1916. The patient returned for the first time since the removal and treatment of the diseased teeth. She

ILLUSTRATING DR. ROWE'S ARTICLE ON "TOXIC CHOROIDITIS."

FIG. 1.
X-ray showing area of bone absorption at apex of central incisor

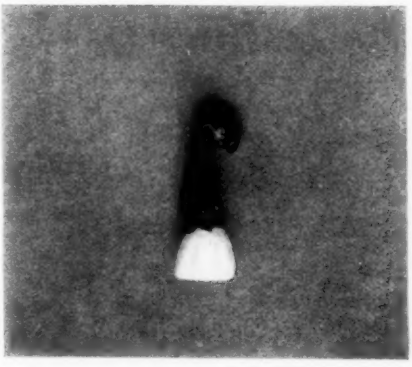

FIG. 2.
Photograph of extracted tooth showing an absorbed apex with adherent granuloma and degeneration of root.

had had no medication save for the local treatment previously mentioned. *Examination of right eye:* Vision $^{20}/_{40}$. Deposits on Descemet's membrane were greatly lessened, and the vitreous opacities were not so numerous. Nerve head and eye ground were quite visible; media was still hazy. *Treatment:* Subcutaneous injection of 1*cc* mixed infection phylacogen, P. D. Co. Atropin and dionin were continued. Strychnia gr. $\frac{1}{30}$ before meals.

Dec. 29, 1916. Vision $^{20}/_{30}$. Few deposits on Descemet's membrane. Disk was clearly visible. The blood-vessels were normal. A large area of yellowish choroidal degeneration was found toward the temporal side of the nerve head. *Treatment:* 1*cc* mixed infection phylacogen. Locally, drops were continued.

Jan. 2, 1917. 1*cc* mixed infection phylacogen, local treatment was continued.

Jan. 6, 1917. 1*cc* mixed phylacogen; other treatment was discontinued. Vision $^{20}/_{32}$. Descemet's membrane clear.

Jan. 10, 1917. Vision $^{20}/_{32}$. *Treatment:* 25 minims mixed phylacogen.

Jan. 12, 1917. 1.5*cc* mixed infection phylacogen.

Jan. 15, 1917. 1.5*cc* mixed infection phylacogen.

Jan. 20, 1917. Wassermann reaction reported negative.

Jan. 24, 1917. S $\frac{20}{24}$. A few opacities in the vitreous remain. The choroidal focus has subsided and shows secondary pigmentation about the usual atrophic area. The disk and retinal vessels are normal. Patient's general health had improved. She had gained twelve pounds in weight. The case was exhibited at Johns Hopkins Dispensary.

In reciting the above case history, I am aware that I offer nothing unusual, either clinically or in the method of treatment. I believe, however, that this will prove of interest to the clinician as well as the ophthalmologist.

It is obvious that the ophthalmologist in order to understand the relationship of dental infections to eye complications, is more or less dependent upon his brother—the dentist, to supply the dental pathology. Black (1), in his recent monograph on dental sepsis and eye diseases, divides the ocular disturbances resulting from dental lesions into two classes—namely, functional or reflex neuroses and organic or inflammatory changes. The former are usually due to an irritation of an exposed vital pulp of a tooth through thermal changes, *i. e.*, heat and cold, or direct contact with foreign substances. We are also reminded that it is possible to have an infected tooth at the same

time that the pulp remains partially vital. The terminal branches of the fifth nerve supplying the teeth are the medium through which reflexes of the motor, sensory, or vasomotor disturbances act, manifesting themselves in various ocular disorders. To this group belong the accommodative anomalies, both complete and partial paralysis, the phorias, spasm of the orbicularis, and cases of vague asthenopic symptoms that are not relieved with optical corrections, and which we are only too prone to call "neurasthenics." Hancock (2) of London, as early as 1858, records a case of strabismus and ptosis disappearing after the removal of abscessed teeth. The second class, the inflammatory or organic, are the result of an infection of an alveolar process or pus pocket along the root. The infection is usually endogenous, occurring through the lymphatics, blood circulation, or by direct continuity. In a review of the literature on this subject, all of the cases reported were of toxic origin; in not one did metastasis occur. Beaumont (3), in quoting Lang's statistics, states that 64% of the cases of iritis of septic origin are caused by pyorrhea alveolaris. Davis (4), in a contribution on the ætiology of choroiditis, cites three case histories of the exudative type in which the source of infection was traced directly to alveolar abscesses, and the exciting organism found to be the staphylococcus pyogenes albus.

While admitting the possibility of the classification just mentioned, and believing as I do that reflex neuroses are as likely to be caused by an irritated tooth as by a deviated septum, yet I am inclined to consider this condition as of infrequent occurrence and to look upon dental infections as most commonly the direct cause of both the functional disorders and the true inflammatory changes. These functional disturbances need not be referred solely to the eye, but may result from general toxic absorption, as was exemplified in our own case. We note our patient had lost weight, appetite was poor, was unable to sleep, and was extremely nervous, a sort of prodrome of some months' standing, which preceded the local eye lesion, and was due to a focal infection.

Treatment: Parke Davis & Co.'s stock vaccines for mixed infections were used in this case. For many reasons it was impossible to use the autogenous vaccine. Although the vision

had greatly improved after the teeth extraction, I feel that the vaccines, while they were begun late, were still in a measure responsible for the happy results obtained. I am aware that vaccine therapy, in local eye diseases, although lauded by some, has been the cause of not a few unfavorable terminations. For that reason, I began the injections of smaller doses than usually prescribed, and at the same time prolonging the intervals. At no time was there either a local or a general reaction.

Summary: In conclusion I wish to emphasize several very interesting facts that I have endeavored to bring out in the study of our case. First, the necessity of localizing and then treating the focal point of infection. Second, the importance of a mouth examination as a routine. Indeed, with the advent of the radiograph in dental work, as well as the laboratory methods at our command, it is to be hoped that the so-called "idiopathic expressions" will soon become obsolete in ophthalmic literature. The ophthalmologist of to-day, when he receives his first warning of a local manifestation of a more remote disease, should include, in his general survey, an examination of the blood, the urine, the accessory sinuses, the mouth and what not. The time is not far distant when we shall see oral surgery an important adjunct not only in the practice of ophthalmology, but also in general medicine, a fact I dare say now recognized by most internists. We need go just one step farther, so important is this branch of medicine becoming, that in the future all hospitals either special or general will be equipped with dental departments.

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ANTERIOR-CHAMBER SYRINGE FOR WASHING OUT CORTICAL MATTER AFTER CATARACT EXTRACTION.

By JOSEPH A. ANDREWS, M.D.

Consulting Ophthalmic Surgeon, City Hospital, New York City.

(*With one illustration in the text.*)

EVERY ophthalmic surgeon, who employs irrigation of the anterior chamber of the eye for the purpose of washing out cortical matter after cataract extraction, must, at some time, have experienced the inconvenience of having his glass tip clogged with a particle of rubber washed off from the interior surface of the bulb. Another objection to allowing the salt solution to come in contact with the rubber bulb is that particles of the rubber surface may be injected into the eye. It is true that these particles are sterile; nevertheless, their presence in the eye is objectionable. The tip of the irrigator shown in the cut is simply the same thing, on a much larger scale, as the writer described in these Archives several years ago.[1] The new model is large enough to contain all the solution necessary for an irrigation without allowing the solution to enter the rubber bulb. The receiver, which has a capacity of two drams, is held obliquely, at the temporal side of the eye, the *flat surface* of the *tip* being *parallel* with the *body* of the *reservoir*, instead of at *right angles* to the

[1] Joseph A. Andrews, "On the Injection of a Weak Sterile Solution of Sodium Chloride into Collapsed Eyes," Archives of Ophthalmology, vol. xxix., No. 1, 1900. Also "An Instrument for Syringing out Cortical Matter in Cataract Extraction," *Transactions of American Ophthalmological Society*, July 19, 1892.

glass tube in common use, and which is operated from above, instead of at the temple. It has been my habit, before using

the rubber bulb, to partly fill it with water (after boiling), to rub its walls together, thus detaching loose particles of rubber, after which the bulb is thoroughly rinsed, before using it for irrigation.

The apparatus is made by F. A. Hardy & Company of Chicago and New York.

THE VALUE OF EYE MANIFESTATIONS COMPLICATING FRACTURED SKULL.[1]

Preliminary Report.

By MARTIN COHEN, MD., New York.

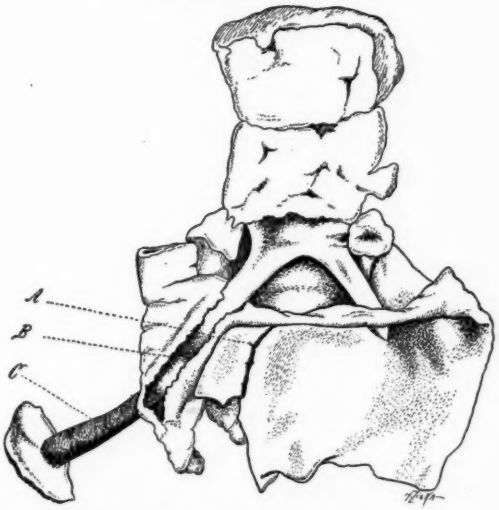
(With two illustrations on Text-Plate VI.)

THE association of ocular lesions and visual disturbances with fractures of the skull is important from the viewpoint of diagnosis, prognosis, treatment, and forensic medicine. In obscure cases an ocular examination may in certain fractures, which tend to recover spontaneously, give a very bad prognosis as to vision, while other severe and extensive fractures, with a very bad prognosis for life, may not lead to serious damage to vision, so that if the patient recovers he may completely regain his vision. In suits for damages, an expert opinion may be required, in order to exclude the possibility of the existence before the accident of a unilateral or bilateral eye lesion resulting from luetic disease, nephritis, arteriosclerosis, serous pachymeningitis, etc.

The literature of the subject is neither extensive nor very satisfactory. A number of surgeons have reported on very large series of cases of fractured skull, without, however, devoting much space to the eye findings; the latter consisting largely of observations on the pupillary changes. Garach, in a Paris thesis, 1914, collected all the cases he could find in the literature, but his notes of thirty-two published cases,

[1] Read at meeting of Ophthalmic Section, New York Academy of Medicine, February 19, 1917.

ILLUSTRATING DR. COHEN'S ARTICLE ON "THE VALUE OF EYE MANIFESTATIONS COMPLICATING FRACTURED SKULL."



A—Line of fracture.
B—Canalicular part of optic nerve covered with blood clot.
C—Disturbed optic nerve sheath covered with blood clot.

Microscopical drawing of above section showing:
A—A sub-dural hemorrhage of the intervaginal space.
B—A distended sub-arachnoid space devoid of hemorrhage.
C—Marked compression of optic nerve fibers.
D—Moderate upward displacement of the lamina cribrosa.

including personal material, have only a limited value because of the incompleteness of some of them, the absence of X-ray findings, the scarcity of autopsy material, and the failure to note end results.

I have records of seventy-five cases of fractured skull, treated at the Harlem Hospital, which were confirmed by X-ray pictures, and by autopsy in fatal cases. In nearly all of these, ophthalmoscopic examinations were made. In nineteen, fundus lesions were present. The type of fracture in this material is very largely that known as indirect basal fracture, such as is due to blows or falls on the cranium. Eye lesions have been known to follow simple contusion, but in the interest of simplicity, they are not included in this present material. In the great majority of fractures, both base and vertex were involved through irradiation, but in practice such cases are all classified under fractured base. Violence may have been suffered in any one of the three fossæ, but naturally fractures into the anterior and middle fossæ are those in which the eyes are most likely to be compromised. It is seldom that fracture, involving the posterior fossa, irradiates into the bony structure in the neighborhood of the visual organs.

It has already been stated that simple contusions of the vertex or head injuries of any kind, outside of fractured base and vertex, are eliminated in this study as causes of eye manifestations. All fractures from bullet wounds of the skull, producing hemianopsia, all cases of direct violence, direct traumatic paralysis of the extrinsic ocular muscles, and pulsating exophthalmos, are also excluded. Our subject is limited therefore to pupillary changes, and to fundal changes, such as venous stasis, papillitis, neuroretinitis, optic atrophy, retinal hemorrhages, and changes in the visual field.

Eye manifestations should be brought in relation with brain injury causing papillitis, such as hemorrhages in orbit, optic sheath, and vicinity resulting from direct trauma or contrecoup, and actual bone injuries to the orbital wall, optic canal, and vicinity, causing laceration or compression of the optic nerve. A study of Garach's thirty-two cases fails to reveal any such mechanism. In some statistics, the orbital wall is involved in two thirds of all cases of fractured base.

In eighty-six cases of fractured base, collected by Von Hoelder, the orbit was involved in ninety-one per cent., and the optic canal in fifty-three per cent.

Certain conditions may antedate the trauma, and may be confounded with changes due to the injury, or may act as contributory causes by offering an element of predisposition. These include congenital anomalies, chronic inflammatory processes, and high refractive errors. Generally speaking, one must bear in mind that the results of trauma must differ as to whether they act upon a healthy or an abnormal eye condition. The most important sequelæ of fractured base, such as pupillary inequality, pseudo-papillitis, and venous stasis, may all be present within a physiological condition in some subjects.

The eye manifestations, following fractured skull, may be regarded as due, in a general way, to one or more of the following morbid conditions: cerebral hemorrhages, due to direct injury or contrecoup, laceration of brain tissue, orbital hemorrhages, increased intracranial pressure, hemorrhage in sheath of optic nerve, and possibly osseous compression or simple severing of the optic nerve. The manifestations may be divided into pupillary and fundal.

Pupillary findings:	Normal
	Unequal
	Bilateral dilatation
	Bilateral contraction
	Light reaction.
Fundus findings:	Mild papillitis
	Moderate papillitis
	Retinal hemorrhages
	Unilateral primary optic atrophy
	Changes antedating the trauma.

Unlike other observers, I have never—during a service at the Harlem Hospital for the past five years, where we average seventy-five cases of fractured skull annually—observed a single case of choked disk—that is, an evident elevation of the papilla. This condition requires time for development, and in fatal cases death occurs too early for its manifestation; while in the recovered cases it never occurred.

In the material under consideration, inequality of the pupils (in the recovered cases) was noted three times. This condition in the dilated pupil was associated with absence of the direct and indirect light reaction, while in the other pupil, the direct reaction was normal, and the indirect was absent. In the dilated pupillary eye in these cases, the fundus remained normal only in the early stage of the condition, from three to six weeks. One of the cases was associated with an immediate and marked unilateral amblyopia, while the other two were accompanied by an immediate unilateral amaurosis. Such cases are important from a visual and medico-legal standpoint, and possibly also from the point of surgical intervention, provided the X-ray establishes the presence of bone displacement; but up to the present time this point has not been verified. Inequality of the pupils, producing a unilateral traumatic dilatation without any apparent optic nerve involvement, may also be present in cases terminating fatally; autopsies in such cases showed cortical hemorrhages, ruptured meningeal artery, lacerated brain tissue, hemorrhage into various portions of the optic nerve sheath, local subdural hemorrhages around the optic canal, local hemorrhages into the retro-orbital tissue, hemorrhages into the accessory sinuses, and fractures of the skull without any apparent bone displacement in the neighborhood of the optic canal.

The question arises whether this traumatic pupillary dilatation can be explained by the existence of cortical centers for visual motor impulses, such centers having been injured by bone compression, localized cerebral hemorrhage, or lacerated brain tissue. This pupillary dilatation generally occurs on the same side as the cerebral hemorrhage.

The general and local symptoms of fractured skull are too well known to need recapitulation. Since in many fractures which cause eye manifestations, the lesion occurs or radiates in close proximity to the eye, we often see subconjunctival chemosis,—hemorrhage into the lid,—which is not due to direct violence to the eyeball, but rather to direct extension from the retro-orbital hemorrhage.

There is naturally great difficulty in conducting an ophthalmological examination soon after the injury, but pupillary conditions are readily noted. These are frequently

normal. The fundus changes might present venous stasis, a mild papillitis, and retinal hemorrhages. As soon as the patient recovers consciousness and ability to orientate himself, his visual acuity may be determined by subjective tests. Cases differ very much in this respect. In one type, the acuteness of vision is at once markedly diminished on one or both sides, even to complete blindness. Thus, after a brief period of coma, the patient may find himself blind in one eye; if an ophthalmoscopic examination can be made at this time, the fundus will probably present nothing unusual. In this type of cases unilateral optic atrophy gradually develops, visual disturbances usually appear immediately, but frequently only within a few days to a few weeks after the injury. This delayed manifestation is not perceived by some patients at first, owing to the fact that the disturbance is unilateral. The amblyopia and the amaurosis are probably due to some interference with the conductivity of impulses affecting directly the visual and pupillary fibers of the optic nerve. Perimetric examination will eventually show various restrictions of the visual field.

In another class of cases, there are relatively slight disturbances of vision after the injury, which are readily explained by slight fundal alterations, such as venous stasis and mild papillitis. In this class, the damaged vision and fundal changes improve and the usual result is complete restoration of vision, with a gradual disappearance of the fundal lesions.

Since a very large proportion of cases of fractured base end fatally, we can only observe the course of the ocular disturbances in the relatively mild cases. Generally speaking, those that recover are likely to suffer from temporary or permanent visual disturbances, one of the most conspicuous results being the unilateral amaurosis or amblyopia. A neuroretinitis or papillitis appearing a few days or weeks after the accident, might indicate the onset of a meningitis, as we have been able to demonstrate by autopsy in three cases.

As regards vision, the cases which show unequal pupils, with no response to direct or indirect light reaction in the dilated pupil, followed later by a descending unilateral primary optic atrophy, result inevitably in a marked and permanent deterioration of the vision. Later the dilated pupil undergoes

a secondary contraction. In all other cases, whether or not there are pupillary or fundal changes, there is apparently ultimate restoration of normal vision, the fundus becoming normal.

The only microscopical autopsy findings in the visual apparatus as illustrated consisted of hemorrhages in the sheath of the optic nerve, mainly its orbital and optic canal portion. This hemorrhage was due to an extension from hemorrhages in the cerebral subarachnoid space, or localized hemorrhage of the optic nerve sheath, subdural hemorrhages in the vicinity of the optic foramen, hemorrhages into the accessory sinuses surrounding the optic canal, fracture in the vicinity of the optic canal,—the canal itself possibly being involved,—and localized retro-orbital hemorrhages of the extrinsic ocular muscle. In performing autopsy on a fractured skull, a careful examination should be made of the entire optic nerve, including the orbital portion, the optic canal portion, and the intracranial portion.

In case of unilateral optic atrophy, all in survivors, we naturally have no anatomical findings, hence we have no absolute data relative to the cause of this lesion. If it could be demonstrated by the X-ray that optic atrophy is produced by displacement of bone pressing upon or severing the optic nerve posterior to the entrance of the central retinal vessels, or possibly if a hæmatoma of the orbital part of the optic nerve sheath could be determined, surgical intervention might be considered as a means of preventing marked amblyopia or amaurosis. Garach notes the frequent occurrence of hæmatoma in the sheath of the optic nerve, only to conclude that it plays no rôle in causing unilateral optic atrophy. A microscopic study of the tissue of the visual apparatus is now being conducted and will be reported later.

There were seventy-five cases of fractured skull studied for this report; twenty-four ended fatally. The cause of death in all but three was cerebral hemorrhage, death occurring in from a few hours to a few days after the injury. The other three deaths were due to meningitis; two died on the sixth day, and one a month after the fracture.

I wish here to express my thanks and appreciation to the attending surgeons at the Harlem Hospital, and also to

Dr. Charles S. Cassasa, for their kind interest and assistance in the work of this preliminary study.

Pupillary findings in 24 fatal cases

Normal	6
Unequal	15
Dilated	3
	24

Light reaction was absent in the majority of cases

Fundal findings in 24 fatal cases

Normal	8
Neuroretinitis	3
Mild papillitis (bilateral)	3
Mild papillitis (unilateral)	1
Moderate papillitis	1
Retinal hemorrhages	2
Chronic chorioretinitis	1
No examination	5
	24

Pupillary findings in 51 non-fatal cases

Normal	44
Unequal	4
Not noted	3
	51

Light reaction was sluggish in the majority of cases.

Fundal findings in 51 non-fatal cases

Normal	35
Unilateral primary atrophy	3
Mild papillitis	6
Not noted	7
	51

Conclusions

1. Inequality of the pupils combined with bilateral absence of the light reflex is very common in fatal cases of fractured skull, while comparatively rare in the cases that recover.

2. Where there is inequality of the pupils associated with a unilateral marked amblyopia or amaurosis in the eye in which

the pupil is dilated, one must recognize the subsequent possibility in this eye of a resulting descending primary optic atrophy.

3. Lesions of the fundus are relatively infrequent, especially in the cases that recover.

4. Papillitis usually indicates the presence of a meningitis, an increase in the cerebral pressure, or a hemorrhage into the sheath of the optic nerve or its immediate vicinity.

5. Contrary to the usual observations, choked disk was not observed in any of these cases of fractured skull.

6. The presence of a unilateral optic atrophy should lead to the investigation of a possible previous head injury, if all other causes are excluded.

7. In the cases terminating fatally, the pupil was generally dilated on the same side as the corresponding cerebral hemorrhage.

8. Fundus examination immediately following skull injuries may be helpful from the medico-legal point of view, as it may give evidence of the preëxistence of contributory factors.

REPORT OF THE TRANSACTIONS OF THE SECTION ON OPHTHALMOLOGY OF THE ROYAL SOCIETY OF MEDICINE.

By Mr. H. DICKINSON, London.

A meeting of this Section was held on the 7th of February, the President, Mr. William Lang, F.R.C.S., in the chair.

Mr. C. Hartridge showed a drawing of a case of **probable rupture of the optic nerve.** The patient was a member of the Royal Flying Corps, who while making a flight had fainted suddenly. He remembered nothing until five days after the event. The machine had been observed to descend, gradually at first, but vertically in the last 500 yards. The machine was smashed up. The patient sustained serious damage to the left arm and right eye; there was no apparent skull injury. Three weeks after the accident there was extensive subconjunctival hemorrhage in both eyes. In the right eye there was no perception of light, the pupil was widely dilated, the vitreous full of blood, so that no details of the fundus could be seen. There was a small wound of the upper lid. A probe did not discover a foreign body. The vitreous hemorrhage gradually became absorbed. The vision in the left eye was $\frac{6}{5}$; fundus normal. The right eye remained blind.

The President spoke of a similar case in which the optic nerve was torn through the boy being poked in the eye with a clothes prop. Half the field only was preserved, the deficiency corresponding to the area involved by the tearing.

Lieut.-Colonel Elliot commented on the man escaping with his life, which was probably due to the impact of the fall having been borne by one of the wings. He recently saw two men who had been in a similar fall from 150 feet up, and one

of them has slight ecchymosis of the conjunctivia, and the other a sprained ankle.

Mr. GRIMSDALE showed a case of **congenital absence of choroid.** The masses of pigment were not heaped up, but apparently lay flat. Vessels were scattered over the greater part of the white sclerotic.

Mr. ARNOLD LAWSON reported in detail three cases of **fur infection of the conjunctiva of children from cats.** The patients belonged to the well-to-do class, and were healthy before the occurrence of the particular infection.

The first case was that of a girl, age 15 years, who was devoted to a Persian cat. One evening, after romping with the cat, she buried her face in its fur. Soon afterwards her eyes became suffused, and she had a severe sneezing attack. On the following day the eyes were swollen, and a discharge appeared. A few hours later the condition had become very acute, the eyes streaming with pus. The nostrils were also much swollen and purulent, and a sanguineous fluid was oozing out of them. Cultures of the cat's fur and of the exudation from the child revealed staphylococcus pyogenes aureus in abundance. Energetic treatment on conventional lines soon effected an improvement, and recovery ensued without permanent damage to the eyes. The cat was destroyed.

The second patient was a boy, æt. 14, and his case was complicated and severe. A fortnight before being seen by Mr. Lawson he had slight inflammation in the left eye, with slight discharge, and swelling of neck glands. The local doctor regarded it as mumps, and advised isolation. It, however, did not behave as mumps, and as the condition became worse, Mr. Lawson was consulted. There had been no fever or pain. He found the whole of the left side of the face swollen, and over the parotid region there was a brawny induration, and the skin was somewhat reddened over the angle of the jaw. The lids of the left eye were so swollen that examination was difficult: and there was a muco-purulent discharge in the furrows of the swollen tissue. Tubercle seemed improbable, on account of the rapidity of the development. The boy had been in the habit of walking about with a pet Persian cat perched on his left shoulder, and cuddling it. The boy was put under a general anæsthetic and the disease treated with

cutting, scraping, and swabbing with 2% nitrate of silver solution. Subsidence, and then disappearance of the symptoms ensued, but it was three months before he was cured. The scrapings showed streptococcus longus, streptococcus brevis, and staphylococcus albus. In the fur of the cat, staphylococcus albus was found, and in its skin staphylococcus citreus and some non-pathogenic varieties. The cat was destroyed.

The worst case was the third, that of a little girl æt. 5 years. When staying with friends near a farm, she played a good deal with a cat which also visited a neighboring cowshed. The child showed muco-purulent conjunctivitis, of mild type. After a time, a number of neck glands became involved, the latter gradually becoming worse. On the upper lid he found an exuberant mass of cockscomb-like granulations on the retro-tarsal tissue and on the posterior edge of the tarsal plate. He suspected it was conjunctival tubercle. Vigorous treatment resulted in an excellent recovery after three operations at various dates. In the intervening four years the child had remained well, and was so still. Mr. Browning pronounced the scrapings to contain tubercle bacilli, probably the bovine type. Scrapings containing only a few bacilli were injected into a guinea-pig, and it rapidly wasted, and was dead within a fortnight.

Mr. Lawson said he narrated these cases because he did not consider that the danger of domestic pets, especially cats, was yet sufficiently realized by the public.

In the ensuing discussion, cases of the conveyance of influenza, tubercle, diphtheria, and scarlet fever by cats and dogs were mentioned.

Mr. P. C. BARDSLEY read a paper entitled: **The retinal signs of arteriosclerosis compared with those due simply to increased blood pressure.** He started with a review of the work on this subject by the late Marcus Gunn, which had not received any material additions since that great effort was published. Gunn's four cardinal retinal signs of arteriosclerosis were: (1) a tendency towards tortuosity of the arteries, especially smaller vessels; (2) a variation in the caliber of the vessels, particularly a sudden diminution over a short length, passing again into the normal size; (3) alteration of the normal

light streak, which becomes brighter and more sharply defined; (4) indentation of veins. Marcus Gunn was one of the first to point out that arteriosclerosis was not confined to old age, and that old age was not alone productive of these changes. The prognosis of these cases was formerly given as very bad, but the present author noticed some years ago that the outlook was not so universally bad, for some advanced cases, with considerable retinal œdema and hemorrhages, improved very much under treatment, and in some the cardinal signs largely disappeared. These changes sometimes occurred with startling rapidity, especially after some toxæmia, such as scarlet fever or influenza: and it was not conceivable that arteriosclerotic changes could occur in a day or two. The patients, as he found from examining many cases, always had a high blood pressure, and often with associated headaches; and when the blood pressure was reduced, the retinal signs of sclerosis appreciably diminished. He maintained that it was possible to distinguish with the ophthalmoscope between the signs of arteriosclerosis and those of simple heightening of blood pressure. The latter condition was associated with the following appearances: (1) The vessels have the appearance of *uniform* distension and fullness; (2) the light streak is *broadened out;* (3) the light streak is larger than normal, and does not take on the appearance of silver wire; (4) the tight arteries indent the veins, and in the case of very high tension, lead to back pressure. He concluded by indicating a number of pitfalls of which the unskilled ophthalmoscopist must be aware.

Mr. HERBERT PARSONS alluded to the recently published work on the same subject by Mr. Foster Moore.

Mr. J. B. LAWFORD said that not very long after Mr. Marcus Gunn arrived at his conclusions, it was evident to him, the speaker, that the prognosis was not, in all cases at all events, as grave as was believed by Mr. Gunn. He saw a number of cases which presented the appearances of the advanced disease, some of them Mr. Gunn's own cases, in which, however, the grave outlook was not justified by subsequent events. He agreed with Mr. Bardsley's paper.

Dr. D. LEIGHTON DAVIES (Cardiff), speaking of the examination of a number of old people in a large infirmary, said that the most constant feature he noticed was not so much an

indentation of the veins, as a redness of the disk, which was always present. There was a certain indistinctness of outline of the arteries, but not amounting to obvious alteration in their caliber. Yet some of the cases had a systolic pressure of over 200*mm* and a diastolic pressure of 140*mm*. He very seldom saw in these cases the classical appearance of "silver-wire" arteries.

Mr. Bardsley briefly replied.

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By LEWIS W. CRIGLER, M.D., Secretary.

GEORGE H. BELL, M.D., CHAIRMAN. MONDAY EVENING, JANUARY 15, 1917.

Dr. Joseph Bruder showed a child six years of age from whose orbit he had removed a **lead pencil.** The child gave a history of having fallen on its face one year previously, causing a swelling over the right eyebrow and a ptosis of the upper lid. Several physicians were consulted, the first of whom made an incision over the swelling. Discharge persisted from that time until Dr. Bruder was consulted in December, 1916. He made a diagnosis of a foreign body in the orbit, opened the wound, and with a probe detected the foreign body, which proved to be a lead pencil broken into numerous pieces.

The wound healed rapidly following the removal of the foreign body.

Dr. Joseph Bruder presented a patient who had had **retrobulbar neuritis following ethmoiditis.** The patient gave a history of sudden obscuration of vision in the right eye following a cold, and a purulent discharge from the right nostril, associated with headache and a dull pain over the right orbit. Vision was reduced to hand movements. There was slight distension of the retinal vessels and the temporal half of the nerve was pale. Transillumination and X-ray both revealed the presence of an ethmoiditis. There were polypi in both nostrils. These were removed and the posterior ethmoidal cells were curetted. Three days subsequently the patient's vision was restored to $\frac{20}{40}$

Dr. W. W. Weeks presented a case of **retinitis proliferans**

in a man age 44. The patient gave a history of poor vision for the past ten years. The trouble began with two separate attacks of retinal hemorrhages, one month apart. His vision in each attack was reduced to light perception, followed by a slow and only partial restoration of sight. His vision in each eye at the present time is $\frac{20}{200}$ with correction, in both eyes. His family history is negative. Examination reveals signs of an old central choroiditis. Narrow bluish-white bands follow the vessels out from the disk and swing forward into the vitreous in both eyes.

His fields of vision for form and color are irregularly contracted.

Dr. ARNOLD KNAPP presented a **case of fracture of the frontal bone** in a boy ten years of age, who had fallen two stories three months ago. There was a distinct defect in the anterior surface of the frontal bone, involving the orbital margin. The eye was pushed down and out and there was a soft circumscribed prominence in the upper and inner part of the orbital opening, corresponding to the frontal sinus. There were no symptoms on the part of the eye and of the brain. This mass, the nature of which seemed obscure, could be reduced on pressure without causing any inconvenience or pain.

The diagnosis seemed to lie between meningocele, hernia of orbital fat, or distension of the lining of one of the accessory sinuses of the nose.

DISCUSSION: Dr. GUTTMAN asked Dr. Knapp what he proposed doing for this child.

Dr. WOLFF said that he thought that the depressed bone was forcing the soft parts downward and forward.

Dr. SCHIRMER too thought the condition one of mechanical pressure, with chronic inflammation. He thought also that there might be a mucocele of the frontal sinus.

Dr. KNAPP presented the photographs of a case of **bilateral glioma** in a child two years of age. The parents state that a year ago they had been advised to have the left eye removed, which advice they had not followed, and now there was a solid mass as large as an adult's fist, projecting directly forward from the left orbit. This was covered by a dried-blood coagulum, and at one place a depression, according to the mother, was the eye.

The picture corresponds to the fungus hematodes which has been described as one of the outcomes of glioma. The vitreous chamber of the other eye was completely filled with gliomatous material. The extension of a glioma, if left to itself, is of interest. Apparently it proceeds back, enlarging the optic nerve sheath, which it then perforates. It also infiltrates the posterior layers of the sclera. From both of these situations it extends into the orbit, producing an exophthalmos. The minute the growth gains the loose tissues of the orbit its progress is remarkably rapid, and the anterior part of the eye, which seems to be in some cases more or less preserved, is pushed forward on the summit of this protruding mass.

DISCUSSION: Dr. SCHIRMER said that it was extremely rare that gliomata ever reach this advanced growth, as the children usually die beforehand.

Dr. GEORGE H. BELL showed a case of **unilateral optic atrophy in a child eight years of age.** The family history was negative and the etiology obscure. Three years ago the child was struck in the face just below the left eye. It was necessary to take three stitches in the wound. Since that time the vision of the left eye has steadily declined and now the patient can detect hand movements at five feet only. There is a simple atrophy of the optic nerve. The right eye is normal.

Dr. BELL thinks there may have been a fracture into the optic foramen, resulting in pressure upon the nerve. X-ray examination revealed slightly cloudy ethmoids. The sella was normal.

DISCUSSION: Dr. KNAPP said that he did not think this a case of simple atrophy due to fracture. He said there were degenerative changes in the retina, especially at the macula, as well as a perivasculitis. He thought a more thorough investigation would reveal some other cause.

Dr. SKEEL said that he thought the condition one of simple atrophy, and that it might have been produced by a fracture.

Dr. BELL agreed that there were some degenerative changes in the retina, but he thought that this condition had developed subsequent to the atrophy.

Dr. LEWIS W. CRIGLER presented a case of **steel in vitreous chamber** three months after injury with **no inflammatory symptoms and retention of useful vision.** There was no

siderosis, the eye was without pain, and vision was $\frac{20}{30}$. The foreign body which was located by means of the X-ray was situated near the posterior margin of the ciliary body; it measured 1 by 3*mm*. The patient was advised to submit to its removal, but refused. He was told that the eye would without doubt be lost in time, and that there was some danger of sympathetic ophthalmia. The physician who attended the patient after the injury failed to detect the presence of a foreign body.

Dr. CRIGLER presented a patient with **an extensive exudation into the deep layers of the retina of the right eye.** The case was that of a young girl, 14 years of age, who gave a history of gradual failure of vision in the right eye for the past two years. Her mother has had four miscarriages, she being the only living child. Her personal history and physical examination throw no light on the subject as to the nature of the disease, although the conformation of her face, a partial deafness in both ears, and her mother's history of four miscarriages (in the face of a negative Wassermann), are strongly suggestive of hereditary lues. She has not the characteristic Hutchinson's teeth.

This patient was seen in September, 1909, by Dr. E. L. Meierhof, from whose record I obtained the following notes:

O. D. V. $\frac{20}{40}$, opaque nerve fibres and chorioretinitis.

O. S. V. $\frac{10}{200}$, not improved by glasses; choroidal areas of atrophy in macular region; marked divergence of left eye.

Examination of the right eye by me on Dec. 7, 1916, revealed the following conditions:

Marked deviation outward, vision reduced to hand movements; no external evidence of inflammation, cornea and aqueous clear, cortical opacities in the posterior lens fibres, large veil-like opacities floating in the vitreous fluid. The disk margins are obliterated and the surrounding retina elevated slightly unevenly as far forward as the equator. Along the anterior border of this elevation in some places are small hemorrhages and one or two ampullated veins. In the periphery of the lower temporal field there is an area of chorioretinitis along margin of which there are other small hemorrhages. The retinal vessels are distinctly seen throughout their course until they reach the periphery, where they are

slightly blurred by the acute swelling in the areas mentioned above. Here they become somewhat tortuous and newly formed vessels are seen. The entire normal choroidal reflex is obliterated by this massive exudation, except the lower nasal part, where a small amount of normal retina has not become involved, and the background presents a silvery white appearance with cholesterine crystals deposited in different parts of the retina.

The left eye has a myopia of 15 D. There are choroidal changes at the posterior pole. Vision with correction is $\frac{20}{50}$.

DISCUSSION: Dr. ARNOLD KNAPP said that this was undoubtedly a case of "Retinal Disease with Massive Exudation" as described by the late Dr. George Coats.

Dr. GEO. W. JEAN agreed with Dr. Knapp.

Dr. H. H. TYSON said that the interesting point to determine in these cases was the etiology. He has had under observation and treatment for the past three years a case quite similar in appearance, in an adult male 42 years of age. He had been unable to determine the cause, notwithstanding all tests were made. The patient's vision is now reduced to light perception in the affected eye. While this condition is supposed to be of a tubercular nature, he is of the opinion that in this case an underlying specific lesion is at least an associate cause. He considers the prognosis poor as regards vision. Increased tension should be combated by iridectomy, and the patient should be treated as though the lesion were syphilitic and tubercular, combined.

Dr. H. H. TYSON reported a case of **electric amblyopia and ophthalmia** following an intense exposure to "broadside arc lamps." On December 3, 1916, Miss D. T., age 20 years, brunette, a moving picture actress, while having a "close up" photograph of her eyes taken, was obliged to gaze steadily for five minutes at a large broadside arc lamp about two feet in length, of 60 amperes and 110 voltage, which was placed at a distance of about two feet from her eyes. In the studio were four lamps, two large broadside arc lamps as just mentioned, and two Hewett lamps, the latter of 30 amperes and 110 voltage each. Immediately after the exposure, upon closing her eyes, she could see bright images of the lights. With her eyes open, she saw a dazzling bright yellow spot which lasted for

about two hours. This was followed by a vertically oval blind spot, about the size of a man's head, which appeared in the center of her visual field. During the evening of the same day she experienced severe headache with itching and burning of eyelids and profuse lacrimation, which increased in intensity during the next day and still persists, one month after the exposure. The absolute scotoma persisted for twenty-four hours. On December 5th, her eyes were slightly suffused and sclera of a pinkish tint. Vision with correction in each eye was $\frac{20}{30}$. There was a relative central scotoma for red and green.

Ophthalmoscopic examination showed œdema of retinæ, especially along the blood-vessels, with blurring of the veins, congestion of the macula region, and hyperæmia of the disks.

Sodium iodide, rest, and euphos glasses were prescribed. On January 5th, vision and color perception were normal, but the eyes became rapidly fatigued. Itching and burning of eyelids continue. Fundi show faint white lines along the vessels.

It is of interest to note that many of the workmen about the studio wear no protecting glasses and experience all the symptoms of mild electric ophthalmia; and there is no doubt but that many of the latter cases are treated in our clinics as ordinary conjunctival catarrh, as a result of carelessness in taking histories. In studying the etiology of eye diseases it is well to bear in mind the possible effect of elctricity as a direct or indirect factor in the case.

GEORGE H. BELL, M.D., CHAIRMAN. MONDAY EVENING, FEBRUARY 19, 1917.

Dr. JOHN E. WEEKS presented **two cases of tuberculosis of the conjunctiva.** The first case was that of a girl nine years of age. He saw her for the first time last October. He said that he was familiar with two types of tuberculosis of the conjunctiva: one acute with miliary tubercles following injury, with rapid growth and involvement of the pre-auricular gland; another type with ulceration of the palpebral conjunctiva of slow chronic type. Inoculation in this variety is either by way of tear duct or by direct extension from the skin adjacent to the eye (lupus vulgaris). The form here presented he has never seen before. Patient was first given a placebo in order that the condition might be watched. The

diseased area involved the upper palpebral conjunctiva and transition fold. There were enlarged follicles which showed little or no tendency to ulceration. The lids were expressed without relief. In January, the second case, four years of age, was seen. It was identical with the first. A piece of tissue was removed from the affected conjunctiva and inoculated into the anterior chamber of a rabbit's eye. A piece was also stained and mounted in order to study the tissue and search for the tubercle bacillus. Giant cells and caseous masses were found, and one acid-fast bacillus was also seen. Three weeks after the rabbit's eye was inoculated there was no growth, but at the present time there are distinct nodules in the iris. In both cases there was pre-auricular gland involvement. The gland was removed from the younger patient for study. It showed the characteristic tubercular degeneration.

Von Pirquet skin reaction was doubtful in the older child, but positive in the younger. No systemic involvement could be determined. Both cases show some improvement following treatment with tuberculin. Dr. Weeks thinks that the infection in these two cases was due to a slight injury to the conjunctiva.

DISCUSSION: Dr. LAMBERT said that he did not recall having seen anything quite like the two cases shown. He had, however, seen a case with a peculiar mass at the sclerocorneal margin which had previously had a tubercular choroiditis. The mass disappeared under tuberculin injections.

Dr. SCHIRMER said that twelve years ago he had seen a young man 20 years of age, who had been bitten by a dog that had just eaten a tuberculous placenta of a cow. A few weeks after the injury, the eyelid which was bitten became inflamed, the pre-auricular and submaxillary glands became involved. The condition was proven to be tubercular by means of animal inoculation. He treated the case by curetting the diseased area with a sharp curette. The affected glands were removed. The patient had a relapse. Tuberculin at that time was not used. Dr. Schirmer described a third form of tuberculosis of the conjunctiva as papillary or cauliflower-like in form. He said that he thought that the treatment of all of these varieties should consist in curetting the affected

parts with the sharp spoon. He said that the prognosis in most cases thus treated was favorable.

Dr. WEEKS said that he thought it best to treat the cases with tuberculin before resorting to the curette on account of the scar formation after the use of the curette.

Dr. OTTO SCHIRMER presented a patient who had been affected with **optical hallucinations.** A full report of this case appears in the ARCHIVES OF OPHTHALMOLOGY, vol. xlvi., No. 3.

DISCUSSION: Dr. WEEKS said that he had reported a case similar to the author's, several years ago. He said that Dr. Schirmer's theory as to the cause was correct, in his opinion. There is an excitation of the cells of the visuo-memory center.

Dr. CRIGLER presented for Dr. CHARLES GRAEF a patient affected with congenital dislocation of both lenses, with loss of useful vision.

DISCUSSION: Dr. LAMBERT advised needling these lenses while the patient was yet young. He said that he had obtained good results in several cases.

Dr. GEO. H. BELL said that the tendency of partially dislocated lenses was to become totally dislocated, and in that event they become cataractous. He thought for that reason, as Dr. Lambert had advised, discission should be performed as early as possible.

Dr. C. E. MCDANNALD presented a case of **vernal catarrh that had received one treatment with radium.** A few days following the treatment the patient developed a severe conjunctivitis, central keratitis, and a dermatitis which lasted for several weeks. Later, keratitis developed in the right eye, which ran a slow course. He thinks the radium was improperly administered.

DISCUSSION: Dr. MAY said that radium was an effective agent for good when properly administered in these cases. He considers 25*mg* the proper dose when it is properly screened.

Dr. MCDANNALD presented a case of **trachoma apparently associated with vernal catarrh, treated with high frequency current.** Dr. McDannald first saw the case in 1912. At that time he found a granulomatous mass situated on the conjunctiva of the right upper lid. It was removed and sent to

the laboratory where a diagnosis of round-cell sarcoma was made. The patient was lost sight of until nine months ago, when both eyes presented the appearance of a mixed infection of trachoma and vernal catarrh, although no eosinophiles were found. The patient had a dense pannus covering the upper half of both corneæ. In spite of all treatment for trachoma including three operations (scarification and expression), the patient grew worse. In desperation he decided to try the use of the high frequency current. The patient received treatment once a week of a minute's duration for the first two weeks, and then at intervals of three weeks from August to December. The patient now shows almost complete recovery, the pannus having disappeared.

DISCUSSION: Drs. MAY and WEEKS thought that it was not unlikely that the case presented a mixed infection.

Dr. MARTIN COHEN read a paper on **The value of eye manifestations complicating fractured skulls; preliminary report.** This paper appears in full in the ARCHIVES OF OPHTHALMOLOGY, vol. xlvi., No. 3.

DISCUSSION: Dr. W. H. STEWART said that from a study of the four cases referred to him for roentgenographic examination, two showed a fracture of the skull beginning in the vertical plate of the frontal bone and extending into the roof of the orbit; one did not show a fracture anteriorly, but on a former examination the bony injury was found well posterior, involving the lambdoidal suture. The fourth case also showed a fracture of the vertical plate of the frontal bone, a fork extending downward into the roof of the orbit. In no case were we able to demonstrate an actual extension into the optic nerve canal. Dr. Stewart thinks that in view of the above findings he is justified in assuming that the immediate unilateral loss of vision is due to injury of the soft parts and not to a fracture of the bone.

Dr. WILLIAM SHARPE said that it was not the fracture that concerned the surgeon most in these cases, but the resultant hemorrhage and intracranial pressure which was responsible for most of the damage. Barring infection he considers the fractured skull a safety valve for the relief of this increased intracranial pressure. He said that the reason choked disks were not seen early following these injuries was

that there was usually a period of shock lasting from four to six hours in which the blood pressure did not equal the intracranial pressure; therefore there would be no intracranial hemorrhage until the period of shock began to subside, when the blood pressure would equal or exceed the intracranial pressure. It is during this period of shock that a great many of the cases die. Surviving this period we then expect the signs of intracranial pressure to be made manifest in the fundus of the eye.

Dr. Sharpe considers X-ray pictures of importance in cases of suspected depressed fracture of the vault; he states that it is rare that fractures of the base are located by this means and that such pictures are of little importance in determining the question of operative interference. He considers the early recognition and relief of increased intracranial pressure the prime requisites to success. Frequent ophthalmoscopic examinations and measurements of the pressure of the cerebrospinal fluid by means of a spinal mercurial manometer are the two most reliable means of determining the onset of this increased pressure.

Dr. M. J. SCHOENBERG said that Dr. Cohen's findings in regard to the pupillary changes were quite unusual; he would therefore like to know whether or not cerebrospinal syphilis had been excluded.

Dr. CHARLES B. CASSASA thought, like the speaker, that choked disk following fracture of the skull was infrequent. He said that often the patient died before there was sufficient time for the swelling to develop, and that in cases that survived the initial shock, rapid disintegration and absorption of the clot took place before the increase in pressure became manifest in the eye. On the other hand, papillitis was observed frequently in patients who fully recovered without operation. That the patients in this series reported by Dr. Cohen had increased intracranial pressure was shown by the pulse, blood pressure, and spinal fluid, and, in fatal cases, by autopsy.

Dr. Cassasa said that fracture of the orbital bones was frequent, especially the orbital plate of the frontal and the lesser wing of the sphenoid. Another relatively frequent finding is a large hemorrhage within the capsule of Tenon without fracture of the orbital fossa.

Dr. J. A. KEARNEY said that it was the custom in Dr. Sharpe's service at the Polyclinic Hospital to examine the eye grounds of every case of fractured skull. The usual early findings consist in a general œdema masking all of the details of the disk. By making daily examinations following injury it is possible to detect early the signs of beginning increased intracranial pressure, at which time operative interference is indicated and most likely to result favorably.

Dr. LAMBERT said that he had recently had under observation a patient who emphasized the importance of ophthalmoscopic examination in all head injuries. The case was that of a man 60 years of age who had fallen on the ice and struck the back of his head. He was carried home, apparently uninjured by the fall. A physician was called who treated him for some time for grippe. His vision finally became affected and Dr. Lambert was called. Finding a bilateral swelling of the nerves (papillitis with retinal hemorrhages) intracranial pressure was suspected. An X-ray examination was made and an extensive fracture of the occipital bone, extending to the foramen magnum, clearly shown. The patient subsequently improved; no operation was done.

Dr. GEORGE H. BELL said that he had recently shown a case of unilateral optic atrophy in a child. There was a history of a blow over the malar bone. X-ray examination did not reveal the evidence of fracture. He thinks it likely that the fracture was present but that the X-ray failed to show it.

Dr. COHEN said that cerebrospinal syphilis had been excluded in the examination of the cases mentioned.

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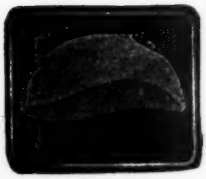

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